Vol. 34, No. 4 August, 1944

Journal of Experimental Psychology

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CONTENTS

Emotional Factors in Experimental Neuroses: M. B. ARNOLD 257

The Effect of Interpolated Activity on Spontaneous Recovery from Experimental Extinction: A. M. LIBERMAN 282

Pitch Discrimination as a Function of Tonal Duration: W. W. TURNBULL ... 302

Studies in Color Blindness: I. Negative After-Images: C. TAYLOR 317

Factors in Massed and Distributed Practice: T. W. COOK 325

The Threshold of Flicker Fusion as a Function of Excitation and Inhibition due to Conditioning: G. K. YACORZYNSKI 335

PUBLISHED BI-MONTHLY.
BY THE AMERICAN PSYCHOLOGICAL ASSOCIATION, INC.
PRINCE AND LEMON STS., LANCASTER, PA.
AND NORTHWESTERN UNIVERSITY, EVANSTON, ILLINOIS

Entered as second-class matter, February 6, 1937, at the post office at Lancaster, Pa., under the Act of March 3, 1879

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THE AMERICAN PSYCHOLOGICAL ASSOCIATION, INC.

NORTHWESTERN UNIVERSITY, EVANSTON, ILLINOIS

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Journal of
Experimental Psychology

VOL. 34, NO. 4 AUGUST, 1944

EMOTIONAL FACTORS IN EXPERIMENTAL NEUROSES

BY MAGDA B. ARNOLD

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The term 'experimental neurosis' has been used to describe an acute or chronic disordered state which appears to be the result either of conflict or of strong stimulation. As it was first found to be associated with conflict (Pavlov, **35**) this disturbance has generally been assumed to be 'emotional' in character, though so far there has been no suggestion as to what emotion could be involved or how a sound-produced behaviour pattern (Humphrey, **17**, **18**; Morgan and Waldman, **31**) could be related to disturbances produced by conflict.

If we peruse the literature on the subject we find that according to the symptoms described and their resemblance to acute emotional disturbances, we could classify the experimental neuroses reported under three headings, *viz.*, chronic fear reactions, chronic rage reactions and explosive reactions.

1. Chronic fear reactions.—This type of neurosis has been described by Pavlov (**35**) as 'inhibitory neurosis' and interpreted as a breakdown of the excitatory process. The animals become unresponsive to positive conditioned stimuli, and the salivary reflex is disturbed or inhibited. In the experimental situation, as well as out of it, the animals exhibit fear reactions (Pavlov's 'defensive reflex'). Symptoms resembling such reactions (trembling, crouching, resistance) have also been described as part of an 'experimental neurosis' by Anderson and Liddell (**1**) in sheep, Karn (**21**) in cats, Maier (**22**) in rats (as 'nervousness'). Very probably, Hall's (**16**) strain of 'emotional' rats belongs in this category.

2. Chronic rage reactions, described so far extensively only by Pavlov (**35**) as 'excitatory neurosis.' In this condition, the animals are reported to become extremely restless, aggressive and agitated. Positive conditioned reflexes become overactive and chaotic, while

negative conditioned reflexes completely disappear. Outside of the experimental situation, the animals remain excitable and aggressive. There is a possibility that the recurring temper tantrum of a monkey, described by Jacobsen *et al.* (**20**) is another instance of this type of upset. Finally, the 'sham rage' described by Bard (**39**) and others may be another example.

3. *Explosive reactions* (after auditory stimulation) reported by Dice (**9**) in mice, by Maier (**22**) and a number of later investigators as occurring in rats. Probably Pavlov's (**35**) description of an explosive motor reaction in one of his dogs during sound stimulation refers to the same phenomenon. Bard (**39**) describes such explosive reactions, also to auditory stimulation, in a decorticated cat.

In this study we shall be primarily concerned with explosive reactions to sound stimulation as observed in rats. The pattern they follow has been exhaustively described in the literature. It consists essentially of sudden attacks of violent running, sometimes merging into tonic and later clonic convulsion with a phase of exhaustion at the end.

It seems certain that, in every case reported, auditory stimulation was necessary to obtain such a seizure. Though this fact seems undisputed, it has been maintained by various investigators that a variety of other factors are responsible for an increase or decrease in the number of attacks elicited. These factors are assumed to be connected either with the stimulating situation or with the psychosomatic condition of the animal.

A. The stimulating situation.

1. *Conflict.*—Maier and Glaser (**23**) as well as Humphrey and Marcuse (**17**) report that conflict significantly increased the number of seizures. This view, however, is challenged by Morgan and Waldman (**31**), who report that five susceptible animals had seizures equally in and outside the conflict situation, while a group of five non-susceptible animals showed no seizures in either case. Unfortunately, the small number of animals in this study forces us to some reserve in accepting this conclusion.

2. *Forcing in the conflict situation.*—Morgan and Waldman (**31**), in the same study, report that forcing the animal to jump by means of an electric shock did not change the number of seizures for the susceptible group and did not produce attacks in non-susceptible animals. However, as it has been shown (Maier, **22**; Bernhardt *et al.*, **4**) that electric shock as such does not produce seizures, this evidence is hardly conclusive. Actually, Maier and Klee (**27**) report that forcing by pushing the animal with a stick does increase the number of seizures.

3. 'Barriers' in the situation.—Maier and Glaser (**23**) found that the attacks increased in number if the rats found themselves confronted by barriers. The more impenetrable the barrier, the more numerous the seizures. In this study, however, only 21 exposures were given, distributed over five different test situations. With such a small number of trials and the well-known variability in susceptibility of the individual rat, chance undoubtedly plays a considerable role, so that it is impossible to take these results very seriously. As a matter of fact, a retest of two different situations showed little reliability.

4. Restraint.—Maier and Glaser (**23**) produced seizures in previously non-susceptible rats by placing small boxes over the animals during sound stimulation, thus greatly restricting their movements.

5. Intensity of stimulation.—Morgan and Waldman (**31**) found that the number of attacks varied inversely with the distance of the rats tested from the source of sound. Humphrey and Marcuse (**18**) report that more seizures were induced with increasing intensity of sound, a finding which is supported by Bernhardt *et al.* (**4**) and seems now well established.

B. Somatic factors.

1. Age and sex.—There has been a suggestion that young animals are more susceptible than adult rats (Maier and Glaser, **26**), which has been confirmed by Farris and Yeakel (**11**). The latter authors have also reported a sex difference in susceptibility to seizures. Their male rats had more seizures than the females in the first 40 days of testing, while the females surpassed the males in susceptibility in the final experimental period (after 125 days). As this is the first time that sex differences have been reported while previous investigators have consistently found that no difference existed, we may be permitted a certain degree of skepticism.

2. Genetic factors.—Maier and Glaser (**24**) found that susceptibility to attacks is inherited, and conclude from their evidence that it is transmitted as a dominant unitary trait. Griffiths (**14**) confirms the inheritance of the sound-produced reaction pattern, but questions its transmission as a single Mendelian dominant. He suggests, rather, the presence of a possible dihybrid factor.

3. Activity.—Humphrey and Marcuse (**18**) report that exercise in the activity wheel either prior to or during stimulation significantly decreases seizures. Bernhardt *et al.* (**4**) observed that less susceptible animals indulged in more motor activity during stimulation (such as climbing, face washing, scratching and biting their body). Maier and Glaser (**25**) found that seizures tend to be inhibited if the animals are subjected to a period of swimming before being exposed to

XUM

auditory stimulation. Long periods (45 min.) in water at 75° F., which caused a definite fall in body temperature, were more effective than shorter periods, or swimming in water at 90° F.

4. Nutrition.

a. Restriction of food and water.—Finger and Schlosberg (**12**), as well as Patton and Karn (**33**), reported that restricted food and water intake and periods of starvation increase the number of seizures in susceptible animals.

b. Vitamin requirements.—Patton (**34**), in a series of careful studies, came to the conclusion that vitamin B_1 deficiency is a contributary cause of susceptibility to seizures.

C. Psychological factors.

1. Handling.—Humphrey and Marcuse (**18**) found that consistent sympathetic handling from birth significantly decreased susceptibility to seizures in rats as compared with a control group of litter mates.

2. 'Emotionality.'—Billingslea (**5, 6**) and Martin and Hall (**29**) report that the F_6 and F_7 generations of a strain of 'emotional' rats were significantly less liable to seizures than the same generation of non-emotional animals. 'Emotionality' in this strain was characterized by timidity, lack of aggressiveness, decreased activity in the open field with increased activity in a familiar situation, and defecation and urination in the experimental situation.

3. Startle.—Turner (**38**) reported that a group of susceptible animals gave startle responses of approximately four times the magnitude observed in normal controls.

4. Accommodation.—There is considerable evidence that accommodation or adaptation both to the bell situation and to handling is fairly rapid, the more so the shorter the time between stimulations. A rest period of two weeks, however, is reported to restore the original susceptibility (Humphrey and Marcuse, **18**). There seem to be some animals (Maier, **22**; Patton, **34**), which show no accommodation at all, while there are wide individual differences in the rate of accommodation in others. For this reason it seems difficult to agree with Farris and Yeakel (**11**), who insist that adaptation is mainly if not exclusively due to increasing age.

D. Drug effects.

In an attempt to investigate whether sound-produced seizures are due to structural or functional factors, several investigators have studied the effects of various drugs.

Maier and Sacks (**28**) found that 23 out of 30 previously non-susceptible animals showed the typical explosive behaviour pattern under the influence of a subconvulsive dose of metrazol.

Humphrey (**41, 42**) obtained an increase in seizures with eserine and nicotine, a decrease with atropine. The nicotine effect, however, was not abolished by atropine, which would prove that events occurring in the parasympathetic system are not a determining cause of seizures.

Finally, Snee *et al.* (**36**) obtained seizures in previously non-susceptible animals after injections of caffeine, metrazol and strychnine in subconvulsive dosage.

Summing up the evidence so far, it would seem that there are a number of factors which contribute to susceptibility, though of such varied, if not contradictory, nature that it is difficult to imagine their relationship. For instance, we know that increased motor activity decreases the number of seizures; yet why should animals of Hall's 'emotional' strain, characterized by *decreased* activity, have fewer seizures than normal controls? And if the sound-produced seizure is an 'emotional' phenomenon, why should there be a direct relation to startle but an indirect relation to 'emotionality'? Furthermore, there is as yet no hint of any relationship between these explosive reactions and the other two types of 'experimental neurosis' reported. And finally, we have just begun to touch upon the relationship of explosive reactions and normal behaviour. Maier's suggestion (**22**) that there is no relation, that these attacks represent a complete break with the normal and therefore are the only reactions that deserve to be called 'abnormal,' has been challenged by Snee *et al.* (**36**), but hardly refuted. Yet this conception of a sharp break between the normal and abnormal contradicts all the knowledge gained in other fields of organismic function. Elsewhere there seems to be a difference in degree rather than in kind between what we agree to be within the normal range and what is considered abnormal.

To illustrate our meaning, we might for a moment turn to one of the other types of experimental neurosis, to what we have called a 'chronic fear reaction.' Here we can trace a gradual transition from normal to abnormal, ranging from transient fear reactions to particular stimuli in the normal animal, through exaggerated fear reactions to any strange situation in Hall's 'emotional' strain, to the pronounced chronic behaviour disorders exhibited by Pavlov's dogs and Anderson and Liddell's sheep. Even within the disturbed group we can follow the gradually increasing intensity of the reaction: some animals seem to 'break down' under comparatively slight provocation, while others have to be subjected to the conflict-producing situation a great many times before showing this change of behaviour, and some do not show it at all. Furthermore, we find that the normal animal quickly adapts to a fear-producing situation. Pro-

XUM

vided no actual harm comes to him, his fear reaction will disappear almost immediately (e.g., in the open field situation), or, at most, within a very few trials. With Hall's 'emotional' strain such adaptation seems to be much slower. In some neurotic animals, as reported by Pavlov, there is also a certain degree of adaptation, though it takes considerable time. If allowed to stay in their living cages, these animals are 'cured' after a more or less prolonged rest. Other animals, however, cannot be cured and, according to Pavlov, show chronic fear reactions for many years. Thus, we could say that the seriousness of the reaction produced varies directly with the intensity or complexity of the precipitating situation (for the animal) and the length of time the animal exhibits these disturbances. It varies inversely with the animal's ability to adjust to emotion-producing stimuli, i.e., with its rate of accommodation.

In sound-produced explosive reactions there is, as in fear reactions, a wide range of individual differences. There are some rats who will react with a seizure every time, while others will have attacks occasionally, and a large group will not react at all. Susceptibility to attack has been found to be genetically transmitted, just as is 'emotionality.' If we can follow the analogy further, we would suspect that it might be possible to find individual animals who exhibit seizures every time to stimuli much reduced in intensity (comparable perhaps to epileptic seizures among human beings).

Despite this apparent continuity, a gap is left between the animals who never show this form of behaviour at all and those that do, however rarely. It could only be bridged if we could show that at least some normal animals possess latent susceptibility which can be demonstrated by increasing the stimulus intensity (either objectively or subjectively) so that an attack will actually be produced. Furthermore, if we could increase the range of susceptibility to the point where a susceptible animal would react to a sound of lower intensity than it has reacted to before, we should bridge the gap between acutely and chronically disordered behaviour.

Finally, having established a similar continuous range in explosive reactions as we have traced in fear reactions, we could then investigate the relationship between these two forms of behaviour.

Thus, our problem is divided into two parts:

1. An attempt to extend the range of susceptibility towards both extremes:
   - *a.* by producing attacks in previously non-susceptible animals, and
   - *b.* by producing attacks in susceptible animals with a stimulus of low intensity.

2. An analysis of the behaviour of normal and susceptible rats during auditory stimulation:
   a. to determine, if possible, the behavioural factors responsible for the attack, and
   b. to investigate their relation to the other two forms of behaviour disturbances.

Problem 1. Extending the Range of Susceptibility

Experimental procedure:

A soundproof box 20 × 20 × 30 in. with a double glass window on one side for observation was used for all trials. In one end, separated by a celluloid sheet from the main compartment, was a bell built on the principle of a rotary hammer fixed at a constant distance from a metal dome. While with a bell of this type the intensity of sound is more constant than with an electric bell, there were some slight variations which it was not possible to exclude. The intensity of sound was controlled by a rheostat which was left in the same position for Experiment 1a, but set at lower intensities for Experiment 1b.

To increase susceptibility we used strychnine, in dosages from .45 mg./kg. upwards. The injections were given subcutaneously in Groups I and II in Experiment 1a, intraperitoneally in Group III, Experiment 1a, and in all three groups in Experiment 1b. Absorption time was empirically determined. Susceptibility was found to be greatest from 20 to 25 min. after subcutaneous injection, from 8 to 12 min. after intraperitoneal injection. Duration of auditory stimulation was two min., both for the preliminary determination of susceptibility and for trials with strychnine.

Experiment 1a. Producing attacks in previously non-susceptible animals by injections of strychnine

This experiment was carried out in three series, consisting of three different groups of albino rats. Groups I and III were rats bred in our own laboratory and handled a great deal, and therefore very tame. Group II consisted of animals brought into the laboratory from outside when almost fully grown, and therefore markedly wilder.

All these rats (N = 33) were tested for susceptibility when fully grown, for 10 consecutive days in Groups I and II, and for six consecutive days in Group III, with the sound at maximal intensity, and were found to be immune to seizures. After two weeks' rest, strychnine injections were started. The minimum dose for Group I was .25 cc. of a .05% solution of strychnine hydrochloride per rat (weight range: 166 to 250 gm.) for Group II. 20 *cc.* per rat of the same solution. This dosage was used as long as an animal continued to exhibit seizures. After at least one trial without seizure, the dosage was increased by .05 cc. per rat. This method, however, was felt to be unsatisfactory because the weight of the rat was not taken into account. Therefore, Group III was started with a minimum dosage of .50 mg./kg., and the dosage increased in the same way after one seizureless trial by .05 mg./kg. With Groups I and II., injections were given daily, with Group III every other day, to minimize drug adaptation. All rats which did not have seizures with lower dosages received dosages up to 1.50 mg./kg. Beyond this we found no more seizures in preliminary experiments, but instead, increased tremor, twitching, and occasionally strychnine convulsions, both with and without bell stimulation. Thus it seems reasonable to suppose that this dosage represents the upper limit for our purposes.

Results:

Out of the above 33 previously non-susceptible rats, 18 had seizures after strychnine injection. These seizures differed in no essential aspect from those observed in normally susceptible animals. The convulsions at the end of the seizures, if present, varied in

XUM

severity from those usually obtained with non-strychninized susceptible animals to opisthotonic strychnine convulsions.

The proportion of animals having attacks was as follows:

	N	Strychnine-susceptible	Percentage
Group I (tame)	8	5	63%
Group II (wild)	12	8	67%
Group III (tame)	13	5	39%
Total of all groups	33	18	54%

Thus, the percentage of animals made susceptible by strychnine is comparable of that to 45% reported by Snee *et al.* (**36**). The difference between tame and wild (Groups II and III) seems to be due to the heterogeneity of stock rather than to a difference in handling, for Groups I (tame) and II (wild) show no significant difference.

The accompanying tables (I and II) show the number of injections given as well as the dosages and seizures obtained. The dosages used in Groups I and II could not be recalculated into mg./kg. because the weight of each rat was recorded only once a week and not before each injection. However, in Group II it can be seen from the initial weight and the end weight that the increase is not enough to account for the large increase in dosage necessary to maintain seizures in some animals (R 21, RL 21, L 22). On the other hand, in two animals which showed numerous seizures (N 6 and RL 23)

TABLE I

NUMBER OF ATTACKS PRODUCED IN GROUPS I AND II OUT OF THE TOTAL NUMBER OF TRIALS, AT THE DOSAGES INDICATED

Rat	Initial weight	Dosages in cc. of .05% strychnine hydrochloride													End weight
		.20	.25	.30	.35	.40	.45	.50	.55	.60	.65	.70	.75	.80	
Group I															
L 5	240 gm.	—	0/1	—	—	—	—	1/1	1/2	2/2	—	—	1/1	—	no record
N 5	250	—	—	—	—	—	—	0/1	—	0/1	—	1/3	0/1	0/1	no record
N 6	230	—	—	—	—	—	—	5/5	—	—	—	—	—	—	no record
R 8	250	—	—	—	—	—	—	1/2	—	—	3/6	—	—	—	no record
L 8	250	—	—	—	—	—	—	0/1	—	0/1	1/1	1/1*	—	—	no record
Group II															
R 21	209 gm.	2/4	—	0/1	0/1	0/1	—	1/2	0/1	2/3	0/1	0/1	—	—	225 gm.
RL 21	225	0/1	—	1/3	—	0/1	—	0/1	0/1	0/1	0/1	3/3*	—	—	240
N 21	240	0/1	—	0/1	—	0/1	—	0/1	—	0/1	—	0/1	2/3	1/1	250
R 22	210	0/1	—	0/1	0/1	0/1	—	1/1*	—	—	—	—	—	—	220
L 22	240	0/1	—	1/3	—	1/2	0/2	0/1	2/3	1/2	—	—	—	—	260
N 22	209	3/4	—	0/1	—	0/1	3/4	2/4	2/2	—	—	—	—	—	230
RL 23	214	0/1	—	11/14	—	2/2	—	—	—	—	—	—	—	—	315
N 23	166	0/1	—	0/1	—	0/1	0/1	2/3	—	0/1	—	2/4	1/3	1/1	265

* Died after seizure.

TABLE II

NUMBER OF ATTACKS PRODUCED IN GROUP III OUT OF THE TOTAL NUMBER OF TRIALS, AT THE DOSAGES INDICATED

Rat	Initial weight	Dosages in mg. per kg.									End weight
		.50	.55	.60	.65	.70	.75	.80	.85	.90	
46	292 gm.	0/1	0/1	0/1	1/2	1/2	0/1	0/1	0/1	0/1	315 gm.
55	355	0/1	0/1	0/1	1/2	1/2	1/2	0/1	0/1	—	380
56	235	13/13	—	—	—	—	—	—	—	—	285
57	235	1/2	0/2	3/4	0/1	0/1	0/1	0/1	—	—	250
59	230	14/14	—	—	—	—	—	—	—	—	280

— = Dosages not given to this animal.

the dosage remained constant while the weight increased. In RL 23 for instance, the dose of .30 cc. at first represents a dosage of .70 mg./kg., but after three seizures, due to an increase in weight from 214 to 270 gm., it represents a dosage of only .55 mg./kg., and finally becomes inadequate as the weight increases to 315 gm. (i.e., .48 mg./kg.). Thus, it would seem that over and above a factor of accommodation which necessitates increased dosage in the majority of animals, the maintenance of seizures in some animals depends on the absolute amount of strychnine present in the body as well as on the amount relative to body weight.

In Group III, as shown in Table II, there is again marked accommodation in some animals (46, 55, 57) and none in others. It seems likely that this accommodation is similar to the adaptation appearing in normally susceptible animals with repeated stimulation. Since according to Goodman and Gilman (**13**) strychnine tolerance does not develop, we may assume that the accommodation observed represents an adaptation to the stimulating situation. And as there is no significant difference in the accommodation shown between wild (Group II) and tame animals (Groups I and III), we would say that it is an adaptation to sound rather than to handling.

From this part of our experiment it seems established that there is a continuous range from previously susceptible to previously non-susceptible animals: with some rats very small dosages produce a great many seizures without appreciable adaptation; with others, the initial dosage required for seizures is higher and has to be increased continually because accommodation is rapid. And finally, some rats, in spite of maximum dosage, do not show seizures at all.

Experiment 1b. Producing attacks in susceptible animals with low intensity stimulus

Seven rats which were found susceptible in 12 trial runs were given intraperitoneal injections of .5 mg./kg. every other day, three times. The rheostat controlling the bell was set at three

positions of varying sound intensity, Low, Medium, and High. (This group of rats had been found to be susceptible to the bell at high intensity, but not to either medium nor low.) After strychnine injection, the rats were tried at the lowest position of the rheostat for one minute; if no attack resulted, they were given 30 sec. rest and then tried at medium intensity for another minute; if there still was no attack they were given another 30 sec. rest and then tried at high intensity. In this way they were within the four minute range of greatest susceptibility (*cf.* p. 263), for all three trials. If a rat had no attacks during any one trial at all, the dosage was increased by .05 mg./kg. As accommodation was very pronounced on the third day of trial, the animals were given a week's rest before the second series of three injections was started. Between the second and third series, the rats had three weeks' vacation to ensure that the strychnine effect had worn off. Before a new series was started, a drugless run (with high intensity sound) was given to determine whether the animals were still susceptible. In the first drugless run (i.e., between the first and second series) three attacks were obtained, in the next run (between the second and third series) four attacks, which corresponds to the general percentage of attacks in this group (30 percent of total number of stimulations).

Results:

In three series of trials, we obtained an increased number of attacks and attacks at lower intensities of sound, as shown in Table III. We notice first of all an increase in attacks from 30 percent in

TABLE III

NUMBER OF SEIZURES PRODUCED IN NORMALLY SUSCEPTIBLE ANIMALS WITH VARYING INTENSITIES OF SOUND, AFTER STRYCHNINE INJECTION

	Intensity			Number of attacks
	Low	Medium	High	
First days, each series	0	11	4	15
Second days, each series	0	2	4	6
Second days, higher dosage	0	2	0	2
Third days, each series	0	2	3	5
Third days, higher dosage	1	1	0	2
Total of attacks	1	18	11	30
Percentage of attacks in 59 strychnine trials	2%	30%	18%	50%
Percentage of attacks in 102 trials without strychnine	0%	0%	30%	30%

drugless trials to 50 percent in strychnine trials. Moreover, of the attacks obtained after strychnine injection, 30 percent were obtained under medium intensity stimulation which without strychnine did not produce any attacks in this group of rats. That increase of dosage increases susceptibility still further is shown by the fact that a dose of .55 mg./kg. produced an attack in one animal with low intensity sound stimulation. Accommodation is again rapid, as is shown by the difference between first, second and third days of injection.

Thus, strychnine has allowed us to extend the range of susceptibility toward normality by producing attacks in previously non-susceptible animals, and towards abnormality by producing more attacks in susceptible animals, and attacks in response to lower

intensities of sound, than was possible before. Where Maier found an absolute break between the normal and the abnormal, we find degrees of susceptibility which may be increased or decreased by varying the animal's physiological condition.

From the fact that accommodation exists in animals made increasingly susceptible, and shows similar individual differences as seen in normally susceptible animals, we find further support for the suggestion that there is a continuous range extending from the normal to the chronically disordered, which seems to show an approximately normal distribution.

Problem 2. Behavioural Analysis

Experiment 2a. Behaviour analysis of normal and susceptible animals (without use of drugs)

The data for this analysis were derived from observations on 78 animals. Of these animals, 44 were specially bred for us and represented the F_2 generation of a cross between white, black and white hooded, black, and agouti. These animals were transferred to our laboratory at the age of three months, and were very wild. The rest were animals bred in our own laboratory and handled consistently, thus were quite tame. They included Group III (N13) of experiment 1a, and the seven albinos of experiment 1b, as well as one litter of young from the above F_2 generation of cross breeds.

Method:

All animals (with the exception of the above 20 animals from Experiments 1a and 1b, of which we already had records) were given a minimum of six exposures on consecutive days. The percentage of animals having attacks was distributed as follows:

Of 44 wild rats (mixed stock) ..... 11,[1] or 25%;
Of 34 tame rats (28 albinos, including 20 from Experiments 1a, 1b, 6 mixed stock) ..... 9, or 26%.

To ascertain whether there was a behavior pattern which would be characteristic of normal and of susceptible animals, we used the time-sample method. During each two-min. period of stimulation, 16 observations were made at 7½-second intervals, describing the animals' behaviour at the time of recording. The following descriptive terms were used:

1. Sniffing (which included all head movements),
2. Moving (exploratory body movements, usually with sniffing),
3. Climbing (elevation of forepaws and body, usually with sniffing),
4. Face washing (includes also scratching, biting, and other manipulatory limb movements),
5. Trembling (tremor without discernible body or limb movement),
6. Twitching (sudden body twitches),
7. Lip-wetting (tongue protrudes with a licking motion),
8. Running (only recorded as such when the speed was obviously greater than in exploratory movements),
9. Jumping (quick standing or running jumps),
10. Backing up (rapid backward movements),
11. Turning (rapid turning about and pivoting),
12. Inactive (when no movements could be discerned),
13. Startle (recorded only if unmistakable; mere changes in position were not counted).

On analysis of the data it was found that several categories could be grouped together. Thus, sniffing, moving, climbing, could be grouped together as 'exploratory movements,' all of which had in common the fact that the rat was attending to something in the environment. In the

[1] One animal died after six consecutive seizures.

same way, face-washing included all other manipulatory movements. Tremor, twitching, and lip-wetting were grouped together as 'involuntary movements.' Startle was not included among these involuntary movements because it could occur only once during stimulation, if at all, while all other types of behaviour could recur repeatedly. Finally, running, jumping, backing-up and turning were combined in one category and called 'symptomatic behaviour' because we found that it usually ushered in an attack. Our final categories were:

1. Exploratory movements,
2. Manipulatory movements,
3. Involuntary movements,
4. Inactivity,
5. Symptomatic behaviour,
6. Startle.

These observations were made up to (but not including) the actual attack. As soon as the typical fast running of the excitatory phase started, the bell was turned off. On attack days, therefore, the observations for susceptible animals are reduced to about 8 or 10 (instead of 16 for the full two-min. period), for the average time is about one min. up to the attack.

In addition to the above observations, we recorded urination and defecation in every trial for every rat (number of times urinated, number of boluses).

Results:

First of all we compared the percentage of behaviour in every one of the above categories for normal rats, with the corresponding behaviour pattern of susceptible rats (on non-attack trials). As 'normal' were considered those rats which had neither had an attack in the preliminary six-day stimulation period nor at a later retest. Among our 'susceptible' rats we included not only the animals which showed the complete pattern (excitatory phase, convulsion and coma) in at least one attack, but also those which showed the excitatory phase without subsequent convulsion or coma. This practice may tend to decrease any existing difference in behaviour pattern between normal and susceptible animals, but does give us the advantage of greater numbers for our susceptible group.

We found the following behaviour pattern among our normal and susceptible group (in percentages, averaged *for the total number of trials*, with a minimum of six trials per rat):

	Expl.	Manip.	Invol.	Inact.	Sympt.	Startle	Def.-urin.
	Percentages of behaviour						Nr. per trial
Normal (N=48).....	71.1	13.6	7.1	5.2	0.5	2.5	2.4
Susceptible (on non-attack trials) N=29.....	54.3	17.9	12.4	10.1	3.0	2.3	2.8
Deviation of Susceptible from Normal.....	−16.8	+ 4.3	+ 5.3	+ 4.9	+2.5	−0.2	+0.4

Applying Fisher's χ^2 test, we find that there are less than 12 chances in one million that these differences in behaviour pattern could have arisen by chance ($\chi^2 = 43.197$).

If we consider that this pattern difference is based on a large number of observations of susceptible rats on *non-attack trials*, it assumes considerable importance because of the possibility of predicting whether a given animal will prove to be susceptible or not.

To investigate the change of pattern which occurs on attack trials in susceptible animals, we excluded all the doubtful cases and recalculated the pattern on the basis of observations on 17 rats in all those trials in which they exhibited excitatory phase, and subsequent coma (with or without convulsions). Recalculated in this way, the pattern shows an increase in the difference between normal and susceptible animals, as we suspected. At the same time, there is a highly significant difference between the behaviour pattern exhibited by susceptible animals on non-attack trials, and that shown by the same animals immediately before an attack:

	Expl.	Manip.	Invol.	Inact.	Sympt.	Startle	Def.-Urin.
	Percentages of behaviour						Nr. per trial
Susceptible (N = 17) non-attack trials.	52.9	19.1	12.0	10.5	3.1	2.4	2.9
Attack trials.....	30.0	5.0	3.5	27.5	30.0	4.0	2.5
Deviation.....	−22.9	−14.1	− 8.5	+17.0	+26.9	+2.6	+0.4

There is less than one chance in a million that these pattern differences could have arisen by chance.

Exploratory behaviour.

If we now compare the three behaviour patterns (for normal, susceptible animals on non-attack days, and susceptible animals in attack trials) in greater detail, we find first of all that in normal animals exploratory behaviour is exhibited in preference to every other form of behaviour. In susceptible animals in non-attack trials, the percentage drops markedly, with a further significant drop on attack days.

Inactivity and symptomatic behaviour.

On the other hand, the percentage of inactivity and symptomatic behaviour rises significantly from normal to susceptible animals, and once more in susceptible animals on attack days. We should almost be justified in maintaining that not only is running, turning, jumping and backing up symptomatic for an attack, but inactivity as well.

Manipulatory and involuntary behaviour.

We find the highest percentage of these two forms of activity in susceptible animals on non-attack days; on attack days we find a very marked drop. Normal rats show *less* manipulatory and involuntary activity than susceptible rats on non-attack days, but decidedly *more* as compared to suggestible animals on attack days. This would suggest that manipulatory and involuntary activity may be the means by which susceptible rats can avoid an attack.

If we add to this the qualitative observation that after a period of immobility or tremor the rat will often shake itself and begin to wash its face vigorously, coming to a dead stop as soon as the bell stops, the possibility gains weight that these forms of behaviour are compensatory reactions to a disturbing stimulus. Such an interpretation would be in line with Humphrey's (**8**) and Bernhardt's (**4**) report that neuromuscular activity will prevent attacks. Tremor, twitching and lip-wetting likewise seem to be a direct though involuntary reaction to the situation, for these phenomena are not observed under normal conditions. Tremor has been reported in the literature as occurring in situations of stress (**1**, **16**, **22**) and has usually been identified as a fear reaction.

We shall leave the implication of these findings for our discussion at the end.

Startle.

There is no significant difference between normal and susceptible animals. The difference between susceptible animals on non-attack days and susceptible animals on attack days is spurious because on attack days observation was carried on only up to the moment of the attack proper, as mentioned before. Thus, there will be approximately half the number of observations in attack trials, as compared with non-attack trials. This does not affect the percentages in other forms of behaviour because they all recur at intervals; but it does affect startle which can only occur at the beginning of the stimulation period and would, therefore, have twice the percentage value in the shorter records. For this reason, startle was recalculated for susceptible animals. We found that out of 67 attacks, 31, or 35 percent were preceded by startle, while out of 249 non-attack trials with susceptible rats, 82 animals or 33 percent showed a record of startle. Thus, startle occurred roughly in one-third of all trials with susceptible animals, whether followed by an attack or not. As the percentage of startle is approximately the same for normal animals as for susceptible animals on attack days, the same ratio should hold good for normals.

It is possible, however, that the extent rather than the incidence of startle would have shown a significant difference between the two groups of animals. This would be suggested by the work of Turner (38), who reported startle responses of significantly greater magnitude for susceptible animals than for normal controls.

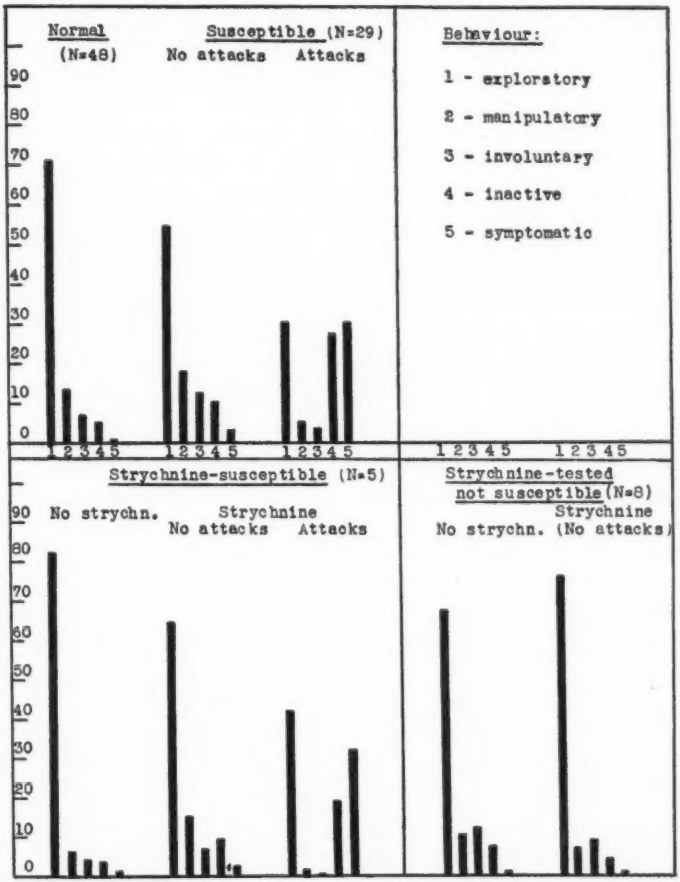

FIG. 1. Percentages of behaviour types in normal, susceptible, strychnine-susceptible, and strychnine-tested not susceptible animals.

Defecation and urination.

There is no reliable difference between normal and susceptible animals in this respect. As defecation and urination have been considered symptoms of 'emotionality' (16) and, more specifically,

a fear reaction, the average number per rat per trial was correlated with the percentages for 'involuntary' behaviour, yielding a coefficient of $+ .39 \pm .065$. Thus, there is a reliable but slight relationship between these two types of fear reaction. In an endeavor to trace the reasons for such a low correlation, we compared the average number of defecations and urinations of tame and of wild animals, and found indeed a reliable difference: tame animals ($N = 34$) averaged 1.8 per trial, while wild animals ($N = 44$) averaged 3.3 ($CR = 4.8$). In this way initial 'tameness' would seem to affect the defecation-urination level of our animals. If this difference in initial level is taken into account by keeping the data separate for tame and for wild animals, we obtain a coefficient of correlation between defecation-urination and involuntary activity of $+ .46 \pm .08$ for wild animals, and $+ .45 \pm .09$ for tame animals. If we can suppose that defecation-urination is a mild form of the same emotional or fear reaction of which tremor, twitching etc. is a more severe symptom, we could reasonably assume that not every animal would show the extreme form to the same degree to which it exhibits a mild form of fear. Moreover, there undoubtedly is a physiological factor and a physiological limit in defecation and urination, which does not operate in tremor.

Experiment 2b. Behaviour analysis for strychninized animals

The 13 animals in Group III, used in our strychnine experiment, 1a, were then compared for their behaviour. First of all, their pattern prior to injection of strychnine was compared with that of normal animals and was found to conform (see Fig. 1). Then their behaviour pattern in trials after strychnine injection, both with and without attacks, was calculated and showed the following deviations:

	Expl.	Manip.	Invol.	Inact.	Sympt.	Startle	Def.-Urin.
	Percentages of behaviour						Nr. per trial
Strychnine-susceptible (N = 5)							
Before injections	82.0	6.0	4.0	3.5	1.0	3.5	1.0
After injection (no attacks)	64.2	15.1	6.4	9.4	2.4	2.5	2.5
After injection (attacks)	41.9	1.4	0.2	18.8	32.1	5.6	1.5
Strychnine-tested, not susceptible (N = 8)							
Before injections	67.3	10.3	12.1	7.5	0.4	2.4	2.7
After injection (no attacks)	76.0	7.2	9.3	4.5	1.3	1.7	0.9

It is important to consider that we are dealing here with *the same*

animals under different conditions of testing, and thus are able to compare their performance under the influence of strychnine and without it with some confidence. Inter-group comparisons, however, (e.g., between strychnine-susceptible and strychnine-tested non-susceptible animals) should be made with due caution.

But it is at least suggestive that we have duplicated in our strychnine-susceptible animals the group differences found previously between normal and susceptible animals, as can be seen from Fig. 1. Without strychnine, their behaviour conforms to that of normal animals, while under the influence of strychnine they approximate the behaviour of susceptible animals both with and without attacks. Without attacks, we find a similar decrease in exploratory movements in the same animals after strychnine injection, as we have found between two different groups (normal and susceptible), with a similar increase in manipulatory and involuntary activity. With attacks, we see similar changes as those occurring in susceptible animals (without strychnine) on attack days.

If we now compare the behaviour changes of strychnine-tested non-susceptible animals, we find that they are in the direction of greater normality after strychnine injection. Instead of a decrease in exploratory behaviour we find an increase, while manipulatory and involuntary activity as well as inactivity decrease.

Discussion

The most important fact gleaned from our data seems to be the inverse relation of seizures to exploratory behaviour on the one hand, and to involuntary and manipulatory activity on the other. All three are accompanied by resistance to seizures; but while a high proportion of exploratory behaviour is accompanied by permanent immunity, involuntary and manipulatory activity seem to be characteristic of a merely temporary immunity in otherwise susceptible animals.

In an attempt to interpret the ratio between these different behaviour patterns, we would suggest provisionally that there may be alternative ways of reacting to auditory stimulation: the nervous excitation set up by the sound stimulus may *either* be discharged immediately and smoothly into a habitual motor pattern (exploratory behaviour), *or* converted into such a pattern with considerable effort (manipulatory movements); *or* it may be discharged via the involuntary (sympathetic) pathway. If such a discharge into either one of these alternative reaction patterns is delayed (inactivity), the excitation will accumulate and finally be discharged into an explosive motor pattern, the sound-induced seizure.

It follows from this hypothesis that we could not expect a direct

relation either of exploratory behaviour to seizures, or of manipulatory and involuntary behaviour to seizures. Of course, the higher the ratio of exploratory behaviour to other types of activity, the greater the probability of permanent immunity. But there may be a marked decrease of exploratory behaviour as compared with the normal, with a corresponding increase in manipulatory and involuntary activity, which combination would also prevent attacks. Thus, a moderate degree of involuntary activity will indicate susceptibility, while an extreme proportion in any one trial will prevent an attack at that time.

It is possible that this hypothesis will resolve the problem as to why it has been found on the one hand that conflict increases susceptibility to attacks (**17**, **22**), presumably by making the animals more 'emotional,' while on the other hand Hall's emotional strain showed significantly fewer seizures than a comparable strain of non-emotional rats. According to our interpretation, the 'emotional' rats converted the sound-induced excitation immediately into a habitual (for them) sympathetic discharge. In a normal group, however, conflict would have the effect of producing excitation without the possibility of an immediate motor outlet; the resultant delay would permit accumulation of such excitation to the point of an explosive reaction. It is significant in this connection that according to Maier's (**22**) explicit statement those rats which showed 'nervousness,' i.e., trembling, squealing, cowering, defecation, and urination in the conflict situation (thus were exhibiting similar symptoms as Hall's emotional strain) did not have attacks.

If we now succeed in identifying beyond reasonable doubt the emotion expressed by these various symptoms, we might also succeed in reformulating our hypothesis somewhat more sharply.

Tremor as well as urination and defecation have long been considered symptoms of fear (**16**, **39**) though non-committal formulations like 'emotionality' (**5**, **6**, **29**) and 'nervousness' (**22**) force us to caution.

Tseng (**37**) has shown recently that defecation, while occurring in the open field situation and (combined with tremor and twitching) under intense sound stimulation, is not found at all in anger situations. (Tseng produced anger reactions by confining one rat at a time in a body-size cage and teasing it with wire for five minutes.) Urination, according to Tseng's report, occurred equally in fear and anger situations. As Hall uses defecation as one of his main indicators of 'emotionality,' this term cannot include an increased liability to anger reactions. Thus the main component if not the only emotion included in the terms 'emotionality' or 'nervousness' would be fear, and the symptoms described under these two terms (decreased

activity, timidity, cowering, squealing, trembling, defecation) could be considered fear reactions. None of these symptoms are found in anger reactions, according to Tseng, but all of them are exhibited by our susceptible rats. In addition, the dry mouth of fear is well known in human experience, parallel to the lip-wetting observed in our experimental animals.

If, then, we agree to label these symptoms fear reactions, we have to conclude that susceptible rats (on non-attack days) show more pronounced fear reactions than non-susceptible animals. On the same premises, we should have to assume that susceptible animals have seizures on attack days because their fear reaction is not strong enough to drain off the excitation induced by the bell; or, in other words, an attack develops before the fear reaction can exert its full effect. This hypothesis is supported by the well-known fact that the overwhelming majority of seizures occurs within the first minute of stimulation. Observing susceptible animals in attack trials we gain the impression that they are overwhelmed rather than afraid. Their 'frozen' immobility, interrupted by short frantic dashes, would suggest that they are unable to convert the excitation experienced into an organized behaviour pattern and are helpless under the impact of the stimulus. It is tempting to draw an analogy with a type of 'shell shock' fairly common in the last war (to-day's 'panic state') which was characterized by blind flight under shell fire with subsequent collapse and amnesia. Another type, characterized by pronounced and incapacitating tremors (to-day's 'battle-neurosis'), could easily be taken as a chronic form of the acute tremor response observed in our susceptible rats. That continuing stimulation does increase the tremor level, even though in our experiment there was no transfer outside the experimental situation, is shown by a comparison of the percentage of tremor in susceptible rats over a period of time. In the first week of stimulation, the average percentage of tremor per rat per trial was 9.7 percent, while two months later it was 12.7 percent in the same rats. Page (**32**) reports that after repeated electrically produced convulsions some of his animals developed chronic tremors, lasting several weeks, together with other neurotic symptoms. It seems conceivable, then, that such symptoms of fear may be cumulative and, in human beings, who can prolong their experience in imagination, may finally become chronic.

If we are justified, then, in supposing involuntary behaviour to be a fear reaction, we could reformulate our hypothesis by saying that in susceptible animals an attack can be avoided:

a. by vigorous activity (manipulatory activity in our experiment, forced exercise in Humphrey's),
b. by a pronounced fear reaction.

In both cases the nervous excitation produced by the stimulus is dissipated before it has reached explosive proportions. On the other hand, the animals in which a high degree of motor activity prevails either are not affected by the stimulus to the same degree and therefore do not need specific defensive reactions (manipulatory and involuntary activity), or are better able to dissipate the excitation the moment it is produced via the motor pathway. In either case, their fear reaction will be less intense than that of susceptible animals.

Experiment 2c

To test our hypothesis, we decided to reproduce the physiological conditions accompanying fear (Cannon, **40**) by the injection of adrenalin. We attempted to keep the dosage as close as possible to the physiological limit, so that it would be comparable to the actual secretion during fear. In preliminary experiments we had found that the lowest dosage which would give tremor effects in normal animals and suppress seizures in susceptible animals was between .003 and .007 mg./kg. Absorption time was found to be approximately one min. To preserve the adrenalin for the duration of the experiment (45 min. for the experimental group) we added one drop of 1/100 NHCl.

Results (Susceptible animals):

With a dose of .007 mg./kg. we suppressed seizures in all but one of seven susceptible rats. For the one animal which had a seizure in spite of the injection of adrenalin, we raised the dosage to .0105 mg./kg. and obtained no more seizures. After 10 days' rest only one of the seven animals had regained its previous susceptibility. Qualitatively, somatic restlessness and muscular tension which had been marked before adrenalin administration in all these rats, was still much reduced.

Behavioural analysis.

We finally calculated the behaviour pattern for our group of seven susceptible animals with and without adrenalin, and found that their pattern was not markedly different from that previously shown in non-attack trials. Inspection of the individual records shows that the increase in exploratory activity is almost wholly due to those animals which before adrenalin administration had exhibited a considerable number of attacks, i.e., had shown violent motor activity. The increasing involuntary behaviour was found in those animals which had not had many seizures. Qualitatively, the change in behaviour of the most susceptible animals was very striking: during the first few seconds of stimulation, they would exhibit all the symptoms of an impending attack, starting to dash ahead; suddenly,

	Expl.	Manip.	Invol.	Inact.	Sympt.	Startle
	Percentages of behaviour					
Susceptible rats on non-attack days (N=7) (without adrenalin).....	54.8	12.6	9.5	16.5	3.0	3.6
Same, with adrenalin.....	58.1	10.5	13.5	12.9	2.7	2.3
Compared with total of susceptible animals on non-attack days (N=34)..	54.3	17.9	12.4	10.1	3.0	2.3

they would begin to twitch and tremble and from then on merely moved around slowly, sniffing, without further sign of 'symptomatic' behaviour. Those animals which were less susceptible and might not have had an attack even without adrenalin, showed noticeably more tremor and less overt movement than before adrenalin injection. Thus, we are justified in maintaining that in every case there was a reduction in gross motor activity. At the same time, the amount of involuntary activity observed in any one rat was not greater than that shown by some rats in previous trials without adrenalin.

It seems pertinent to mention here that with larger dosages we obtained more pronounced symptoms in preliminary experiments, though here again the effects were found duplicated in some of the animals without adrenalin. Thus, by prolonged bell stimulation without adrenalin (four min. and more) we could induce complete prostration in some rats. These animals at first seemed weak, dragging their hindlegs, then became completely flaccid, lying prone with hindlegs stretched out. This condition could be duplicated with an injection of .04 mg./kg. adrenalin in rats which before had not shown it, during the regular two-min. stimulation period.

Thus we can say, first, that the effect of small doses of adrenalin is most like the fear reaction observed previously ('involuntary' behaviour) in the most 'normal' animals. While animals which are highly susceptible merely show a reduction of gross activity from 'symptomatic' to 'exploratory' movements, our least susceptible animals show an increasing amount of tremor and twitching. Secondly, effects are more pronounced with larger dosages; within our range, no reversal effect was observed. As the adrenalin symptoms observed have also been found in animals stimulated without adrenalin, it seems reasonable to assume that the dosages used were close to the physiological limit of adrenalin secretion within the conditions of our experiment.

Experiment 2d

To ascertain whether similar results could be obtained with strychnine-susceptible animals, we selected three rats (N6, 56, 59, in Tables I and II) which had had seizures every time, one with a dosage of one mg./kg., the other two with .5 mg./kg. strychnine. Injections of strychnine

were given, as in Experiment 1a, Group III, every other day. On Mondays, Wednesdays and Fridays, the strychnine injection was followed after seven min. absorption time by an adrenalin injection; on the days in between, strychnine alone was given. If, we argued, these animals had attacks on days when they received strychnine only, and no attacks on days when strychnine plus adrenalin were given, we could be sure of the adrenalin effect.

Results (Strychnine-susceptible animals):

The lowest dose of adrenalin which prevented seizures in rats 56 and 59 was .014 mg./kg., with .5 mg./kg. strychnine; for rat N6, .06 mg./kg. adrenalin with one mg./kg. strychnine. With rats 56 and 59, the adrenalin dosage had to be raised to .021 mg./kg. to maintain inhibition of attacks, while rat N6 remained without seizures on .06 mg./kg. adrenalin. On days when no adrenalin was given, seizures were obtained on two successive occasions; after that, no more seizures could be obtained, either with the same or a higher dose of strychnine. The procedure was repeated with rats 56 and 59, after two months' rest had restored their original strychnine susceptibility. Again, after a few adrenalin injections, no more seizures were observed.

If we consider that up to the time adrenalin injections were started all three rats had seizures after strychnine injection every time without increase of dosage (*cf.* Tables I and II), and thus showed no accommodation, it is perhaps reasonable to suggest that the suddenly appearing accommodation was due to the cumulative effect of adrenalin, especially as the original susceptibility was restored in rats 56 and 59 (rat N6 had died) after two months' rest. Such a cumulative effect of adrenalin has also been observed by Anderson *et al.* (**2**) and may be comparable to the increase in tremor level with stimulation over long periods, mentioned above.

Qualitatively, the muscular tension so noticeable in strychninized animals completely disappeared within one min. after adrenalin administration, and the rats became often more flaccid than they had been before strychnine injection. This flaccidity was the more pronounced the more adrenalin was used.

Behavioural analysis.

The behaviour pattern of rats 56 and 59 is given for the sake of interest:

	Expl.	Manip.	Invol.	Inact.	Sympt.	Startle
	Percentages of behaviour					
Strychnine-susceptible rats (56, 59) before drug administr.	75.0	4.0	5.0	6.5	3.5	6.0
Same, with strychnine and adrenalin	37.5	24.0	16.0	19.0	3.0	0

While the changes in behaviour after the administration of both drugs are in the same direction as those observed in susceptible animals on non-attack days, they are so drastic that we hesitate to draw any conclusions before a larger sample becomes available.

Summing up our findings under Problem 2, we conclude that not only intense fear (accompanied by adrenalin secretion) but also adrenalin injections, whether within the accepted physiological rate of secretion or in excess of it, will prevent seizures. Adrenalin injections will also prevent seizures in animals made susceptible by previous strychnine injections.

Summary and Conclusions

This investigation was designed:

1. to test the hypothesis that the sound-produced seizure represents the extreme of a continuous range from the 'normal' to the 'abnormal' reaction to sound, and
2. to determine the factors responsible for sound-produced seizures by a behavioural analysis (time-sample method), and to investigate their relation to other types of behaviour disturbances, e.g., acute and chronic fear reactions.

Strychnine injections (in dosages of .45 mg./kg. to 1.5 mg./kg.) induced explosive reactions to sound in previously not susceptible rats, and increased the susceptibility of animals which had had seizures before so that they reacted with an attack more frequently, and also reacted to lower intensities of sound. Accommodation was found to be similar to that observed in animals which were found to have attacks without strychnine. We conclude from these results that there seems to be a continuous range from the 'normal' animal to the animal made susceptible by strychnine injections, to the susceptible animal which shows accommodation, and finally the susceptible animal with a permanently 'abnormal' behaviour pattern.

The behavioural analysis of the reaction pattern of animals during sound stimulation established a highly significant difference between normal and susceptible animals, and in susceptible animals between behaviour in trials which resulted in an attack and behaviour in those which did not.

The reactions which were prevalent in 'normal' animals as compared to susceptible animals consisted in vigorous bodily activity ('manipulatory' and 'exploratory' behaviour). It was suggested, therefore, in conformity with previous work that such activity represents a defensive reaction to the disturbing situation.

Susceptible animals showed a preponderance of 'involuntary' activity (tremor, twitching, lip-wetting) in non-attack trials, and a

reduction of this type of behaviour (below the percentage found in normal animals) immediately before an attack. Adrenalin injections duplicated the symptoms of 'involuntary' activity in animals which had not shown such behaviour before, and prevented seizures in susceptible rats with dosages from .007 mg./kg. to .0105 mg./kg. In animals made susceptible with strychnine, subsequent adrenalin injections (dosage from .014 mg./kg. to .06 mg./kg.) also prevented attacks. After identifying the 'involuntary' activity of this study with 'fear behaviour' as reported in previous studies, it was suggested that attacks can be avoided by an intense fear reaction accompanied by adrenalin secretion, as well as by adrenalin injection.

Thus, the sound-produced seizure could be considered as a short-circuit of excitation which occurs when neither deliberate motor activity nor an intense fear reaction offer alternative pathways of energy discharge. As sound-produced seizures and fear reactions thus seem alternative, if not antagonistic, behaviour patterns, we should expect that either acute or chronic fear reactions will reduce susceptibility to seizures.

(Manuscript received February 29, 1944)

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THE EFFECT OF INTERPOLATED ACTIVITY ON SPONTANEOUS RECOVERY FROM EXPERIMENTAL EXTINCTION [1]

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Introduction

Theories concerning the nature of experimental extinction have frequently been centered in the assumption that extinction is only a special case of learning. Wendt (**13**), for example, has suggested that extinction is the result of interference from other responses which are acquired or strengthened during the extinction procedure. Winsor (**15**), Hunter (**9**), and, more recently, Miller and Dollard (**11**) have believed that extinction might best be regarded as a process in which the animal learns to stop responding. The justification for either of these points of view is limited, inasmuch as there are several experimentally demonstrable differences between extinction and learning. In terms of an analysis by Hilgard and Marquis (**7**), these differences are indicated by the following findings: (1) that certain drugs have differential effects on the two processes; (2) that negative correlations obtain between the rates at which extinction and conditioning proceed; and (3) that spontaneous recovery repeatedly occurs as a unique sequel to the extinction procedure. A learning interpretation of extinction is not necessarily undermined by the evidence relating to the differential effect of drugs or to the negative correlations between rates of acquisition. In the face of these difficulties one may yet be free to assume that extinction is a kind of negative learning (learning not to respond) which differs in certain more or less insignificant respects from its positive counterpart. The fact of spontaneous recovery, on the other hand, points toward a basic incompatibility with learning theory which can not so easily be dismissed.

It is a signal characteristic of experimental extinction that the extinguished response often returns to a level approximating its full

[1] This paper is the substance of a dissertation presented in September, 1942 to the Faculty of Yale University in candidacy for the degree of Doctor of Philosophy. I am deeply indebted to Dr. Donald G. Marquis under whose direction this research was carried out, and I wish to thank Drs. Clark L. Hull, Mark A. May, Carl I. Hovland, and Irvin L. Child for many suggestions and criticisms.

An abridgment of this paper was presented at the 1943 meeting of the Eastern Psychological Association in New York City.

strength after a short period of rest. This is spontaneous recovery, and it is this phenomenon which most seriously embarrasses any attempt to develop the assumption that extinction is a representative form of the learning process. The nature of the difficulty is readily appreciated upon consideration of one of the most important implications of the learning interpretation of experimental extinction. If experimental extinction is to be conceived as a set of learned responses, then spontaneous recovery can only be the forgetting of those responses. Learned responses, however, are not forgotten with the rapidity and spontaneity which mark the forgetting of extinction. A conditioned response, for example, is likely to be retained for a long period of time; indeed, it is safe to say that it will be retained until it is interfered with by other responses.

In view of this striking difference in retention between extinction and learning it has sometimes been concluded (**7, 8**) that these two phenomena do not properly belong in the same category. A separate process is assumed to underlie extinction, and it is postulated as an underivable characteristic of that process that its extinctive effects are spontaneously dissipated with the passage of time. Such an assumption is undesirable for several reasons: specifically, because it makes difficult an explanation of those cases in which spontaneous recovery occurs to various and sometimes inconsiderable extents,[2] and, generally, because it materially increases the burden of our postulates.

It will be the purpose of this paper, then, to outline and support an hypothesis in terms of which spontaneous recovery may be derived from accepted principles of learning without the assumption of a non-learning process which spontaneously dissipates. Let it be assumed, first, that extinction is a procedure in the course of which an animal learns to stop responding. This may be called negative learning. The particular hypothesis with which this paper will deal involves primarily an attempt to demonstrate that spontaneous recovery is the forgetting of that negative learning.

Spontaneous recovery and forgetting.—It may be significant that the forgetting of learned responses was once assumed to occur spontaneously in time, just as it is now thought that recovery from experimental extinction is spontaneous. The Law of Disuse was formulated on the basis of the frequent observation that forgetting occurred when practice was discontinued. Forgetting took place over an interval of time; it was believed, therefore, that it took place

[2] Hilgard and Humphreys (6) have found that there is no appreciable recovery of a response which has been extinguished by a discrimination procedure. Pavlov (12, p. 123), also, has reported that as differentiation between a positive and negative stimulus proceeds, spontaneous recovery becomes progressively less complete.

because of time. But then it began to be observed that time and metabolism were probably wholly incidental to another and more significant variable. That variable was the activity which intervened between the original learning and the attempted recall. It appeared that the interpolated learning interacted with the originally learned response with the result that the one or the other or both exhibited a decrement. The decrement which is produced by this procedure has been called retroactive inhibition, and retroactive inhibition is now thought to be an almost sufficient condition for forgetting.

It is possible that spontaneous recovery also fits this paradigm—that spontaneous recovery occurs because activity which is interpolated between extinction and test for recovery retroactively inhibits the tendency not to respond. An example may help to clarify the form which this analogy would take. An animal has been conditioned to respond with an eyeblink whenever a 1000-cycle tone stimulus is presented. The experimenter extinguishes this response by repeatedly eliciting the conditioned eyeblink in the absence of the unconditioned or reinforcing stimulus. In the course of a five-minute period following the last extinction trial, several reinforcements are applied to a salivation response which had previously been conditioned to a 5000-cycle tone. It is conceivable that the strengthening of the salivation response—the further reinforcement of the tendency to respond to the tone—will interfere with (retroactively inhibit) the recently established tendency not to respond to the tone. This interference with the extinction will result in the reappearance of the conditioned eyeblink upon the subsequent presentation of the proper tone stimulus.

Under conditions which are as yet inadequately defined, an interpolated activity transfers positively (rather than negatively, as in the case of retroactive inhibition) to previously learned conditioned or verbal responses. It may be expected, therefore, that an interpolated activity will, under special conditions, transfer positively to a previously learned extinction. In terms of the example presented in the preceding paragraph, the extinction of the eyeblink would very likely have been furthered had the interpolated activity taken the form of extinction of the salivation response. The conditions under which interpolated activity will result in positive or negative transfer to a previously established extinction may be formulated generally as follows. The retention of extinction should be facilitated whenever the extinction of the same or a different response is carried out during the interval after extinction. The retention of the extinction should be inhibited whenever an activity is strengthened (reinforced) during the retention interval.

The factor of similarity.—The possibility that spontaneous recovery may be reduced to the terms of retroactive inhibition will, of course, gain support by the demonstration that the retroactive inhibition of extinction does service to the same principles which apply to the retroactive inhibition of learned verbal or conditioned responses. One of the most important of these principles has reference to the factor of similarity. It has long been recognized that the amount of retroactive inhibition which will be generated depends largely upon the similarity between the original and interpolated activities (10). Within certain limits the retroactive inhibition is greater in amount as the similarity between original and interpolated activities is greater. In much the same manner the similarity between the original and interpolated activities should govern the extent to which the extinction will be retroactively inhibited. The amount of inhibition should be maximal when the reinforcement is carried out on the same response which was originally extinguished, and should become progressively less as the interpolated activity is less similar to the originally extinguished response. The extent to which extinction may be facilitated should vary also with the similarity between the original and interpolated activities, from maximal facilitation when the original and interpolated extinction activities are identical, to minimal facilitation when they are highly dissimilar.

A problem arises in the measurement of the similarity relationships among the responses which may become involved in the extinction situation. For verbal materials the evaluation of similarity is largely a matter of *a priori* judgment. It is usually the case that a group of presumably competent individuals assesses the various verbal stimuli and responses in the light of their formal and meaningful similarities. On the basis of these judgments predictions are then made as to the amount of inhibition which will result from the learning of these materials. It should be possible to make equally *a priori* evaluations of similarity in any rather artificial extinction situation in which two or more interpolated activities differed from each other only in respect of the nature of the stimulus. Suppose, for example, that an originally extinguished response is an eyeblink which had been conditioned to a blue light. One would judge that an interpolated salivation response conditioned to a green light bears a greater similarity to the originally extinguished stimulus-response unit than does an interpolated salivation response conditioned to the word 'food.' In the more nearly real circumstance, however, one must analyze a great number of grossly heterogeneous stimuli and responses. For such a situation *a priori* judgments can hardly be reliable. It would be difficult to arrange according to their respective similarities a group of activities which might include blinking to a

tactile stimulus, finger withdrawal to a visual stimulus, and startle to an auditory stimulus. Yet all of these stimulus-response complexes might have been affected to varying degrees by the extinction of another response, and they would, therefore, have become important for possible interference with that extinction (see *The rates of forgetting of conditioning and extinction*, below). In order to evaluate the similarity relationships among these activities it will be necessary to employ some direct functional measure of similarity. Such a measure may be provided by the generalization of the extinction.

It has been pointed out by Hilgard and Marquis (7) and by Gibson (4) that generalization of the conditioned response offers a basis for an empirical measurement of similarity. This measure suggests itself because it is apparent that the stimuli or responses which range along the generalization continuum are related to each other in a fashion which is significant for the ordinary *a priori* definition of similarity. The stimuli on the continuum of stimulus generalization, for example, are similar insofar as they evoke the same or similar responses. The generalization of the extinctive tendency to stimuli or to stimulus-response units [3] other than those directly involved in the extinction indicates a relationship among the stimuli and responses which might also be called 'similarity.' It may be reasoned that the response of not responding is common to all the activities to which the extinction generalizes. To that extent the organism is responding as if there were a similarity.

These considerations dictate, for extinction, a definition of similarity in terms of the relative amount of generalization of the extinctive tendency from the one response to the other. In accordance with this definition, predictions concerning the amount of retroactive inhibition of negative learning would be based on the extent to which the original extinction had generalized to the interpolated activity which is subsequently strengthened. The greater the initial transfer of the extinction, the greater should be the subsequent interference with the extinction.

The rates of forgetting of conditioning and extinction.—The foundations for the analogy of spontaneous recovery with retroactive

[3] The term 'generalization' applies prototypically to the situation in which a number of stimuli evoke the same response (stimulus generalization), or to the situation in which a single stimulus evokes any one of several responses (response generalization). The concept has sometimes been expanded to include the positive transfer of conditioning or extinction, as in the case of the depression of one response following the extinction of another. For molar consideration this latter usage is justified, inasmuch as stimulus generalization, response generalization, and any combination of the two are only special cases of positive transfer. Every instance of generalization reduces to the demonstration that training on one S-R permits the conditioning or extinction of the same or a similar response in fewer trials than would have been required had the initial S-R not been set up. The transfer of extinctive tendencies is a logical extension of the most basic kind of sensory generalization.

inhibition will have been established if it can be shown that interpolated activity has an effect on spontaneous recovery, and, further, that the degree of the effect varies with the similarity between the original and interpolated activities. This demonstration would make plausible the identification of spontaneous recovery as the retroactive inhibition of the learned tendency not to respond. It would not, however, provide a ready explanation of the contrast between conditioning and extinction with regard to the widely different rates at which these two types of learning are forgotten. For a possible solution of this problem it is necessary to inquire into the circumstances under which an interpolated activity can interfere with learned responses of the verbal learning or conditioning varieties.

In order that an activity may successfully interfere with the retention of a previously learned response there must have existed between the original and interpolated activities some functional relationship. In reference to the retroactive inhibition of learned verbal responses this relationship has been called 'similarity.' The retroactive inhibition of one verbal response can occur only to the extent that the interpolated activity has some element of similarity (and antagonism) to the originally learned material. The absence of any such relationship presumably rules out the possibility of retroactive inhibition. A conditioned response will be forgotten insofar as a response which is antagonistic to the conditioned response becomes conditioned to a stimulus which is similar to the conditioned stimulus.[4] Thus, the forgetting of the conditioned response over an interval of time hinges upon the availability of similar stimuli and antagonistic responses. Similarity may best be measured in this case by generalization along a stimulus continuum, or by generalization in the broader sense of positive transfer to a similar response (see *The factor of similarity*, above). By this conception the number of available similar stimuli is determined by the breadth or extent of the generalization. There is no evidence that stimulus generalization of the conditioned response extends beyond the limits of the modality of the conditioned stimulus. Generalization to other responses, if it occurs at all, is not extensive. With regard to the possibility of setting up antagonistic responses it can only be said that relatively few of the responses which are employed in the conditioning experiment are directly antagonistic to each other; most of these responses can coexist with a minimum of mutual interference. It follows from the assumptions (1) that the generalization is restricted and

[4] The conditions underlying negative transfer of the conditioned response are decidedly more complex than is implied in the scheme which has been outlined here. A discussion of these complexities would not, however, materially further this preliminary presentation of the suggestion that extinction is forgotten in compliance with the rules which govern the forgetting of conditioning.

(2) that the antagonistic responses are scarce that only a very small proportion of the normal activities of waking and sleeping life can interfere with the retention of the conditioned response. As a consequence, the conditioned response is likely to be retained with little loss over comparatively long periods of time. This likelihod is, indeed, a matter of fact. Results from numerous experiments (7) indicate that laboratory-established conditioned responses are typically quite resistant to forgetting.

Extinction presents appropriately similar and contrasting circumstances. Like the conditioned response, the tendency not to respond will be retroactively inhibited by an antagonistic tendency which has been set up with reference to an activity which was included within the wings of the generalization gradients of the original extinction, i.e., an activity which is functionally similar to the extinguished activity. In contrast to the conditioned response, however, the extinctive tendency appears to generalize broadly, encompassing a wide variety of stimulus-response units.[5] In this way there is set up a great store of activities which, by virtue of the relationship implicit in the generalization, have a potentiality for inhibiting the tendency not to respond.

There are several indications in the literature that the generalization of extinction is extremely broad—that as a consequence of extinguishing one response, many other responses are depressed to a greater or lesser extent. Pavlov (12) has emphasized in several contexts that the extinction of one response produces a marked general tendency to cease responding. He reports (p. 54):

> A circumstance of especial interest is that experimental extinction of any single conditioned reflex results, not only in a weakening of that particular conditioned reflex which is directly subjected to the extinction (primary extinction), but also in a weakening of other conditioned reflexes which were not directly subjected to extinction (secondary extinction). This latter phenomenon involves not only those conditioned reflexes which were based upon a common unconditioned reflex with the primarily extinguished one (homogeneous conditioned reflexes), but also those which were based upon a different unconditioned reflex (heterogeneous conditioned reflexes). Sometimes secondary extinction reaches a profound degree, involving even the unconditioned reflexes.

That the generalization of positive conditioning is less extensive than the generalization of extinction is to a certain extent indicated by the fact that there is no term 'secondary conditioning' corresponding to 'secondary extinction.'

Williams (14) has recently found that the extinction of a vertical bar-pressing habit generalized to a horizontal bar-pressing habit even though there had been no evidence of generalization of the

[5] The normally extensive generalization of the extinctive tendency may be reasonably explained on the basis of an analysis of the relative extents to which any instances of positive and negative learning involve the setting up of discriminations. This analysis and its implications will be elaborated in a paper to be published soon.

positive habit. This result rather clearly suggests that the extinctive tendency generalizes more broadly than does the corresponding positive learning. Graham's (**5**) results give further support to this conclusion. In a study of the transfer of conditioned inhibition and conditioned excitation from one habit to another, she found that only the conditioned inhibition appeared to transfer. That is, the response of not responding transferred from one situation to another, whereas the positive response did not.

The extensive generalization of the extinction makes it highly probable that the extinctive tendency will be interfered with in a relatively short time. Let it be assumed that the extinction of a conditioned response generalizes to a great number of other responses, conditioned and reflex, causing them to be reduced in amplitude. In any interval following extinction some of these secondarily extinguished responses will occur. It is very likely that these responses will be reinforced—at least they will not become further adapted or extinguished [6]—and they will be restored to their previous level of strength. The strengthening (relearning) of these secondarily extinguished responses must generalize back to the originally extinguished response and interfere with the retention of its extinction.[7] Spontaneous recovery will occur insofar as a great many of the secondarily extinguished responses operate and are reinforced during the interval following extinction.

The probability that an extinctive tendency will be forgotten may be still further increased by the seeming ubiquity of response tendencies which are antagonistic to the extinction. In one sense, any tendency to respond is directly antagonistic to a tendency not to respond. The strengthening of the tendency to respond in activities which had been depressed as a result of the extinction of another response must interfere categorically with the retention of the extinction.

These considerations provide a possible explanation of the fact that extinction is usually more rapidly forgotten than is the corresponding conditioning. The relatively broad generalization of the

[6] It is a legitimate assumption that well-established learned (or unlearned) responses are reinforced upon the greater number of their occurrences. If this were not the case, animals would soon become entirely quiescent.

[7] The assumption has been made that the conditioning (positive learning) of a response does not transfer to the majority of those responses which would be affected by an extinction procedure carried out on that same response. It is fair to ask, therefore, why the strengthening (reconditioning or positive learning, if you will) of a secondarily extinguished response should transfer back to the originally extinguished response. In answer to this question it must be emphasized that the indirect strengthening of the initially extinguished response operates entirely within the bounds of the extinctive tendency, in that it tends to effect only the *restoration* of the extinguished response to its previous level of response potentiality. The interim re-establishment of a secondarily extinguished response cannot carry the primarily extinguished response beyond its pre-extinction level of strength.

extinction and the omnipresence of antagonistic responses will create a circumstance in which the extinction will be interfered with by a great many of the activities which normally occur in the life of the organism. These same normally occurring activities will not interfere with the retention of the conditioned response because the generalization of the conditioned response is relatively restricted and the number of responses antagonistic to it are few. It follows for a specific situation that a given interpolated activity will have a greater effect on the retention of extinction than it will have on the retention of the conditioned response.

The hypothesis which has been outlined above accounts in a reasonable fashion for several special manifestations of spontaneous recovery. Hilgard and Humphreys (6) have found that spontaneous recovery does not occur to any large extent in a response which has been extinguished in the course of setting up a discrimination between one stimulus and another. Pavlov (12, p. 123) has reported that spontaneous recovery becomes progressively less as differentiation between a positive and negative stimulus proceeds. These results fit well into the hypothesis presented in this paper. By the terms of this hypothesis, the primary cause of spontaneous recovery is the normally extensive generalization of the extinctive tendency. The procedure of discrimination learning must serve to constrict the generalization of the extinction, inasmuch as the animal is taught alternately to respond to one stimulus and not to respond to another. As a result there will be little interference with or spontaneous recovery of the extinction.

From the account of spontaneous recovery which has been presented here it would be predicted that there will be less or no spontaneous recovery following *any* procedure which tends to delimit the generalization of the extinction. There are many specific procedures which should narrow the generalization and thus reduce spontaneous recovery. For example, successive extinctions of a response or distribution of trials in extinction should operate much like a discrimination procedure to isolate the extinguished response and so minimize the possibility of interference from other activities.

It has been suggested in this paper that spontaneous recovery is the forgetting or retroactive inhibition of experimental extinction. The experiments to be reported in succeeding sections were designed to provide opportunities for disproof of the most critical implications of such an hypothesis. Those implications are contained in the following propositions.

1. That it is possible to augment or depress spontaneous recovery from experimental extinction by interpolating activity in the interval ordinarily allowed for spontaneous recovery. The renewed conditioning of a second response during the recovery interval should cause a greater amount of recovery of the extinguished response than would have occurred during a rest interval of equal length. The extinction of a second response during the recovery interval should cause less recovery of the extinguished response than would have occurred during a rest interval of comparable length.

2. That the degree of the effect on spontaneous recovery varies with the similarity between the originally extinguished and the interpolated activities.

3. That the operation of a particular activity (in this case, the conditioning of a response) has less effect on the retention of a

previously conditioned response than it has on the retention of the extinction of that same response.

Procedure

Experiment I

The following procedure was designed to determine whether or not the degree of spontaneous recovery is affected by the interpolation of activity between the last extinction trial and the first test for spontaneous recovery.

Group A (15 *Ss*)

Step 1. Conditioning of the galvanic skin response.—A galvanic skin response (Tarchanoff) was conditioned to a 1000-cycle tone. A single break shock (unconditioned stimulus) regularly followed the conditioned tone stimulus after an interval of six sec. The tone sounded continuously until the shock was delivered. The tone-shock combination was presented at intervals which were varied randomly from 30 to 50 sec. Each *S* received 12 presentations of tone-shock in this initial conditioning procedure.

Step 2. Conditioning of the eyeblink.—Within 30 sec. after the twelfth tone-shock trial, the conditioning of an eyeblink to a 60-cycle buzz was begun. For this conditioning, a puff of air, delivered to the cornea, served as the unconditioned stimulus. The buzz preceded the air puff by 450 millisec., and sounded continuously until the puff was delivered. Each *S* received 10 successive presentations of buzz-puff. The intervals between trials varied from 15 to 35 sec.

Step 3. Extinction of the galvanic skin response (1st extinction series).—The conditioned galvanic skin response was then partially extinguished by presenting the tone without the subsequent shock reinforcement for six trials. The first extinction trial followed within 30 sec. after the last (15th) trial of the eyeblink conditioning. The succession of non-reinforced tones was spaced randomly at intervals which varied from 30 to 50 sec.

Step 4. Rest interval.—The subject was allowed to rest for 10 min. following the last trial of the extinction series.

Step 5. Test for spontaneous recovery (2nd extinction series).—At the end of the rest interval the conditioned tone stimulus was again presented six times without the shock reinforcement, and the galvanic skin response was recorded. The presentation interval varied between 30 and 50 sec. This procedure affords a measure of spontaneous recovery from the effects of the first extinction.

Group B (15 *Ss*)

The procedure for Group B was identical with that for Group A, except that the last five min. of the 10-min. rest interval (Step 4, Group A, above) were taken up with a renewed conditioning of the eyeblink. There were 10 presentations of the buzz-puff combination in this five-min. period.

Group C (15 *Ss*)

The procedure for Group C was identical with that for Group A, except that the last five min. of the 10-min. rest interval (Step 4, Group A, above) were taken up with an extinction of the conditioned eyeblink. There were 10 presentations of the unreinforced buzz in this five-min. period.

Experiment II

Experiment II was designed to measure the effect of the interpolated activity on spontaneous recovery as a function of the similarity between the original and interpolated activities.

The procedure for Experiment II was identical with that for Experiment I in all details but one. In Experiment I the conditioned stimulus to the eyeblink was a 60-cycle buzz. In Experiment II the conditioned stimulus to the eyeblink was the onset of a red light. The red light is judged *a priori* to be less similar to the tone stimulus for the galvanic skin response than is the 60-cycle buzz.

Experiment III

The hypothesis of spontaneous recovery with which this study is concerned specified that the interpolation of a particular activity would have less effect on the retention of a previously conditioned response than it would have on the retention of the extinction of that same response

(see *The rates of forgetting of conditioning and extinction*, above). Experiments I and II provide measures of the effect of an interpolated learning (reconditioning of an eyeblink Group B, Step 4, Experiment I) on the retention (spontaneous recovery, Step 5) of the extinction of a response. Experiment III is designed to provide a necessary basis for estimating the effect of this same interpolated learning on the retention of the conditioned response itself.

In the initial training stages of Experiments I and II the conditioning of an eyeblink (Step 2) was interpolated between the previous conditioning of a galvanic skin response (Step 1) and a test for retention of the galvanic skin response (extinction series, Step 3). In Experiment III this procedure is duplicated, except that there is no interpolated conditioning of the eyeblink. Following the conditioning of the galvanic skin response, the *S* is allowed to rest for the five min. usually consumed by the 10 trials of eyeblink conditioning in Step 2. By comparison with this control it will be possible to determine the extent to which the conditioning of the eyeblink in Step 2 affected the retention of the conditioned response.

Summary of experimental procedure.

EXPERIMENT I

Group A (15 *Ss*)
- Step 1. GSR conditioned to a tone—12 trials
- Step 2. Eyeblink conditioned to a buzz—10 trials
- Step 3. GSR extinguished—six trials (1st extinction series)
- Step 4. Rest—10 min.
- Step 5. GSR extinguished—six trials (2nd extinction series)

Group B (15 *Ss*)
- Step 1. GSR conditioned to a tone—12 trials
- Step 2. Eyeblink conditioned to a buzz—10 trials
- Step 3. GSR extinguished—six trials (1st extinction series)
- Step 4. Rest—five min.
  Eyeblink conditioned to a buzz—10 trials
- Step 5. GSR extinguished—six trials (2nd extinction series)

Group C (15 *Ss*)
- Step 1. GSR conditioned to a tone—12 trials
- Step 2. Eyeblink conditioned to a buzz—10 trials
- Step 3. GSR extinguished—six trials (1st extinction series)
- Step 4. Rest—five min.
  Eyeblink extinguished—10 trials
- Step 5. GSR extinguished—six trials (2nd extinction series)

EXPERIMENT II

Group A (15 *Ss*)
- Step 1. GSR conditioned to a tone—12 trials
- Step 2. Eyeblink conditioned to a light—10 trials
- Step 3. GSR extinguished—six trials (1st extinction series)
- Step 4. Rest—10 min.
- Step 5. GSR extinguished—six trials (2nd extinction series)

Group B (15 *Ss*)
- Step 1. GSR conditioned to a tone—12 trials
- Step 2. Eyeblink conditioned to a light—10 trials
- Step 3. GSR extinguished—six trials (1st extinction series)
- Step 4. Rest—five min.
  Eyeblink conditioned to a light—10 trials
- Step 5. GSR extinguished—six trials (2nd extinction series)

Group C (15 *Ss*)
- Step 1. GSR conditioned to a tone—12 trials
- Step 2. Eyeblink conditioned to a light—10 trials
- Step 3. GSR extinguished—six trials (1st extinction series)
- Step 4. Rest—five min.
  Eyeblink extinguished—10 trials
- Step 5. GSR extinguished—six trials (2nd extinction series)

EXPERIMENT III

Step 1. GSR conditioned to a tone—12 trials
Step 2. Rest—five min.
Step 3. GSR extinguished—six trials

Instructions, subjects, and procedure with Ss.—The *S* entered the experimental room and was seated with approximate reference to the head-rest and electrodes (see *Apparatus*, below). The *E* began to apply the electrode paste and the electrodes, and, as he did so, he gave the *S* the following instructions.

"We are interested in studying responses to two different types of stimulation. In the course of the experiment you will receive a number of mild electric shocks. We will adjust the intensity of these shocks before we begin the experiment. Starting with a shock which you will barely feel, we will gradually increase the intensity until you report that the shock has become definitely unpleasant but not painful."

The *S* was then given the test series of break shocks. The intensity of the shock was gradually increased until the *S* reported that the shock had become 'definitely unpleasant.' Assurance was given the *S* that at no time during the experiment would he receive a shock stronger than the one which he had judged to be unpleasant.

While the glass tube (for the air puff) was being adjusted, the instructions were continued as follows.

"You will occasionally receive from this tube a puff of air directed against the eye. The air puff will be just strong enough to cause you to blink. Do not try to keep from blinking to the puff of air. On the other hand, do not keep your eyes closed all of the time. Fixate your eyes on this black cross. Keep them open as you normally would, and blink them as you normally would."

Several trial puffs were delivered to the *S*.

In every case the *S* was asked to remove his head from the head-rest during the 10- and five-min. rest intervals (see Step 4, above). He was told that he would be permitted to rest for a few moments, and was instructed to relax.

The changes from galvanic conditioning to eyeblink conditioning and from eyeblink conditioning to galvanic conditioning were made without warning to the *S*.

The experimental procedure proper, from the first trial through the last, consumed approximately 25 min. The *S* was in the experimental room for instructions, adjustments, and experimental procedure a total of about 40 min.

Undergraduate students in Yale College were *S*s in this experiment. There were 120 *S*s in all. The data from 10 of these *S*s could not, for one reason or another (see *Interpretation of records*, below), be included in the final analysis. With the exception of 25 volunteers, all of the *S*s were paid. The volunteer *S*s were distributed randomly among all the experimental conditions. Each *S* was warned before he agreed to serve in the experiment that he would be given electric shocks. The *S*s were almost entirely naive with regard to the nature and purpose of the experiment.

APPARATUS

Galvanic skin response.—The 1000-cycle tone stimulus was produced by a T. R. McElroy audio oscillator, and was heard by the *S* through a pair of headphones. This tone was 55 db. above threshold. A single break shock from a Cambridge inductorium served as the unconditioned stimulus. The shock was delivered to the back of the right hand through a small copper contact which was coated with Cambridge electrode paste. The neutral electrode was a 100 sq. cm. area of phosphor-bronze on which the palm of the right hand rested. The timing of the conditioned and unconditioned stimuli was controlled by a 1 r.p.m. Telechron motor so arranged as to make and break electrical circuits at the proper intervals.

Silver electrodes, smeared with Cambridge electrode jelly, were secured by means of an Ace bandage to the palm and dorsum of the *S*s left hand. The electrodes were connected across the input of a Burr-Lane-Nims microvoltmeter (1). A potentiometer in series with this circuit was used to buck the greater part of the subject-electrode potential, and so keep the recording instrument on scale. The galvanic skin response was recorded by a General Electric photo-electric ink writing microammeter connected across the output of the microvoltmeter. The occurrence of all stimuli was indicated on the same record by means of a signal marker.

XUM

Eyeblink.—The *S* sat in a straight-backed chair, his head partly enclosed in a wooden box which served effectively to shield him from whatever light was present in the room. The chin was supported on a cushioned block, and the forehead rested against a padded frame. At the far end of the shielding box was an eight cm. square piece of milk glass on which was painted a black cross. This cross was fixated by the *S* while the experiment was in progress.

The conditioned buzz stimulus for the eyeblink in Experiment I was produced by a 60-cycle alternating current stepped down by a transformer and delivered to the *S*'s headphones. This sound was approximately 60 db. above threshold. The conditioned stimulus for the eyeblink in Experiment II was the sudden illumination of the milk-glass stimulus patch by a painted red lamp in position behind the stimulus patch. An electromagnetically operated drop-shutter interposed between the continuously glowing lamp and the milk-glass made it possible abruptly to illuminate and dim the stimulus patch.

The unconditioned stimulus for the eyeblink in Experiments I and II was a puff of air delivered to the lower part of the cornea. For each trial a pressure of 15 cm. of mercury was built up in a glass U-tube and maintained by an electromagnetically held wedge which pinched off the outlet of the U-tube. The release of this wedge caused a jet of air to be expelled through the glass tip against the cornea.

The spacing of the stimuli for the conditioning of the eyeblink was controlled by a large pendulum. In its excursion this pendulum made and broke the various electrical contacts which, through relays, set off the stimuli.

The eyeblink responses were recorded photographically. A light bamboo rod, pivoted at one end, was attached to the eyelid with adhesive at its free end. This rod bore a small spherical mirror at the fulcrum. The light from a single filament lamp was reflected from this mirror onto the vertical slit of a camera. All movements of the bamboo rod were thus recorded on the light sensitive bromide paper which moved past the camera slit. A time reference was provided by the shadows cast on the slit by a toothed wheel which interrupted a wash light 50 times per sec. The incidence of the conditioned stimuli for the eyeblink (buzz in Experiment I, light in Experiment II) was recorded by the shadow of a signal marker in position before the camera slit. The unconditioned stimulus (puff of air) was marked on the record by the shadow of a paper flap which was made to vibrate by the jet of air.

Interpretation of Records

When endosomatic galvanic skin responses are recorded from two active electrodes, complications arise out of the fact that separate and often opposite potentials may be picked up by the two electrodes (3). With bipolar recording, therefore, the general form, latency, and amplitude of the recorded response is necessarily determined by the relative magnitudes and phase relationships of these sometimes antagonistic potentials. This factor has made it difficult to interpret the records yielded by the experiment which is being reported here. For example, it was often the case that the direction of the galvanometer deflection would suddenly reverse in the course of the experiment. Even more frequent was the appearance of a diphasic response. In order to insure consistency in the reading of the records it was necessary to adopt definitive criteria for response and response magnitude. After considerable trial and error the following rules were chosen.

1. To be counted as a response, the deflection must have begun not more than 12 sec. after the onset of the conditioned tone stimulus.
2. The magnitude of the response was calculated with reference to a base line which was defined as the average standing potential during the six sec. immediately preceding the beginning of the response.
3. In those cases in which the deflection was first in one direction and then in the other, the larger of the two responses was counted. The magnitude of this response was calculated from the base line which obtained before either phase of the response began.

It was necessary to discard the results from 10 of the *S*s for the following reasons.

1. Three *S*s did not show any evidence of conditioning during the initial training procedure.
2. The base lines of endosomatic potential for two *S*s were so continuously changing as to make any response indistinguishable from the normal fluctuation.
3. In seeking to make slight adjustments in the headphones during the rest period, five *S*s inadvertently touched the headphone lead with the grounded hand and received a relatively

severe and unexpected electric shock. This shock may have had a considerable and entirely unwanted effect on the recovery from extinction which was then in process.

All response magnitudes are expressed as millivolts.

Results

The effect of interpolated activity on spontaneous recovery.—In Table I are the mean response amplitudes of the galvanic skin

Table I

Mean Response Amplitudes (in Millivolts) on the First Two Trials of the First and Second Extinction Series of Experiments I and II

Experiment I

Group	1st extinction	2nd extinction
A (Rest)	5.30	2.67
B (Interpolated conditioning)	5.47	5.02
C (Interpolated extinction)	5.47	2.00

Experiment II

Group	1st extinction	2nd extinction
A (Rest)	4.95	2.70
B (Interpolated conditioning)	5.42	4.07
C (Interpolated extinction)	5.57	2.62

response for each experimental group in Experiments I and II. These values were derived by averaging the response amplitudes of all Ss on the first and second trials [6,7] of either extinction series.

The first extinction series immediately precedes, and the second extinction series immediately follows, the interpolation of (1) a 10-min. rest for Group A, (2) a five-min. rest followed by 10 trials of eyeblink reconditioning for Group B, and (3) a five-min. rest followed by 10 trials of eyeblink extinction for Group C. Experiment I differs from Experiment II only in that the conditioned stimulus for the interpolated eyeblink in Experiment I was a 60-cycle buzz,

[6] It was considered advisable to lump the first and second trials of the extinction series into a single score because (1) the means of these two trials show less variability than do the first or second trial scores taken separately, and (2) because the first trial of the second extinction series is quite probably subject to disinhibitory-like influences consequent upon the sudden re-introduction of the conditioned stimulus for the galvanic skin response. In consideration of the latter factor it may be assumed that the first response of the second extinction series has less significance for spontaneous recovery, insofar as spontaneous recovery may be distinguished from disinhibition, startle, *etc.*

[7] Response values for the last two trials of the first extinction series cannot provide a meaningful base-line for the calculation of spontaneous recovery. The difficulty arises out of the following two limitations on the significance of these two responses: (1) the responses on the last two trials were frequently so low in magnitude and so ambiguous in form that accurate measurement of them was almost impossible; (2) a zero magnitude on the last two extinction trials does not reflect the possibility that certain subjects may have been extinguished below zero.

The mean response amplitude on the last two trials of the first extinction series (all experimental groups) was 1.63 mv. Comparison of this figure with the mean response amplitudes for the first two trials of the first extinction series, and with the mean response amplitudes for the first two trials of the second extinction series, makes it clear that the procedures of this experiment were quite adequate to produce experimental extinction and spontaneous recovery.

whereas the analogous stimulus in Experiment II was the onset of a light.

The initial comparability of the six groups is seen by comparing the mean response amplitudes of the several groups on the first extinction series (Table I). There is no indication from any comparisons which may be made among these means that the groups differ significantly from each other with regard to amplitude of response before the introduction of the experimental variables. The most nearly reliable difference between any two of these means yields a probability of 30 chances in 100 that a difference as great as the one obtained (and in the same direction) could have arisen by chance.

The effects of interpolated activity on recovery of the galvanic skin response are apparent in comparisons among the mean response amplitudes in the second extinction series for the three experimental groups (Table I). In Table II the mean differences between experi-

TABLE II

COMPARISON OF EXPERIMENTAL GROUPS BY PAIRS IN TERMS OF MEAN DIFFERENCES IN RESPONSE AMPLITUDE ON THE FIRST TWO TRIALS OF THE SECOND EXTINCTION SERIES

Experiment I			Experiment II		
Groups	Mean Differences	P [a]	Groups	Mean Differences	P
B–A	2.35	<.04	B–A	1.37	<.15
B–C	3.02	<.01	B–C	1.45	<.15
C–A	−.67	<.15	C–A	−.08	<.30

mental groups are presented, together with the probabilities that differences as large as those obtained could have arisen by chance.

It is evident that the amount of recovery from experimental extinction which follows the interpolated *conditioning* of an eyeblink (Group B, Experiment I) is significantly greater than the amount of recovery which follows the interpolated *extinction* of an eyeblink (Group C, Experiment I). Although the reliabilities of the other differences are low, it is notable that the differences appear in the same direction in both experiments, and the means are in every case consistent with the hypothesis that spontaneous recovery is affected by the interpolation of conditioning and extinction procedures. The amount of recovery of the galvanic skin response is increased over control-rest (Group A) if the *S* undergoes a reconditioning of an eyeblink response during the recovery interval (Group B). The amount of recovery of the galvanic skin response is decreased as

[a] The figures under the column 'P' have reference to the probability that differences as great and in the same direction as those obtained could have arisen by chance. These probabilities were derived in accordance with the method prescribed by Fisher (2).

compared with control-rest (Group A) if the *S* undergoes an extinction of an eyeblink response during the recovery interval (Group C).

The effect on spontaneous recovery of degree of similarity between original and interpolated activities.—In Experiments I and II a 1000-cycle tone served as the conditioned stimulus to the originally conditioned galvanic skin response. The conditioned stimulus to the interpolated eyeblink conditioning was a 60-cycle buzz in Experiment I; the analogous stimulus in Experiment II was the onset of a red light. Of these two stimuli to the interpolated response, the 60-cycle buzz of Experiment I is judged to be the more similar to the conditioned tone stimulus for the galvanic skin response.

The effect on spontaneous recovery of these two degrees of similarity may be gauged from the means of Table I. It is apparent from the data contained in that table that the interpolated conditioning and extinction procedures of Experiment I had a greater effect on spontaneous recovery than did the analogous procedures of Experiment II. The differences between comparable means of Experiments I and II are all far below any accepted criterion of significance. Yet it must be considered that the mean response amplitudes are, without exception, in accordance with the hypothesis that the interpolated activity which is the more similar to the originally extinguished activity should have the greater effect on retention of the extinction.

The effect of the interpolated activity on the retention of the conditioned response.—The hypothesis of spontaneous recovery to which this study pertains ascribed the short retention of extinction to the possibility that a given interpolated activity will have a greater effect on the retention of extinction than it will have on the retention of the conditioned response (see *The rates of forgetting of conditioning and extinction*, above). It has been found in Experiments I and II that the interpolated conditioning of an eyeblink interferes with the retention of the *extinction* of a galvanic skin response. The procedure of Experiment III provides a control in terms of which it will be possible to estimate the effect of the interpolated eyeblink on the retention of the previous *conditioning* of the galvanic skin response.

In Experiments I and II the first three steps of the procedure, antecedent to the imposition of the experimental conditions, included (1) the conditioning of a galvanic skin response, (2) the conditioning of an eyeblink response, and (3) the extinction of the galvanic skin response (first extinction series). The procedure of Experiment III differed from the first three steps of Experiments I and II only in that there was no interpolated conditioning of the eyeblink response. In Experiment III the *S* was allowed to rest during the five-min. interval otherwise consumed in conditioning the eyeblink. The

effect of the interpolated eyeblink conditioning on the retention of the conditioned galvanic skin response will be reflected as a difference in first extinction series response amplitudes between Experiments I and II on the one hand and Experiment III on the other.

The mean response amplitude for the first two trials of the first and only extinction series in Experiment III was 5.45 mv. This value is to be compared with the mean response amplitudes of the first two trials of the first extinction in Experiments I and II, *viz.*, 5.30, 5.47, 5.47, 4.95, 5.42, and 5.57 mv. (Table I). The most nearly reliable difference between the mean response amplitude for Experiment III and a corresponding mean for Experiments I or II yields a probability of 40 chances in 100 that a difference as great and in the same direction as the one obtained could have arisen by chance. It may be concluded, then, that the interpolated conditioning of the eyeblink had no measurable effect on the retention of the previously conditioned galvanic skin response.

Analysis of the data in terms of recovery ratio.—It becomes possible to make unqualifiedly direct and immediate comparisons among the groups in Experiments I and II when the response magnitudes of the second extinction series are expressed as ratios of the corresponding values of the first extinction series. By this method of analysis the extent of spontaneous recovery is given in a single score, and differences among the groups with regard to initial level of responding (first extinction series) are completely ironed out.

The 'mean recovery ratios' in Table III indicate the amount of spontaneous recovery for each of the groups in Experiments I and II. This table is analogous to Table I.

TABLE III

MEAN RESPONSE AMPLITUDES OF FIRST TWO TRIALS OF FIRST EXTINCTION SERIES EXPRESSED AS RATIOS OF MEAN RESPONSE AMPLITUDES OF FIRST TWO TRIALS OF SECOND EXTINCTION SERIES

Group	Mean Recovery Ratio Experiment I	Experiment II
A (Rest)	.53	.52
B (Interpolated conditioning)	1.37	.79
C (Interpolated extinction)	.38	.47

The mean recovery ratios contained in Table III were computed as follows. For each *S* the total response amplitude of the first and second trials of the second extinction series was calculated as a ratio of the total response amplitude of the first and second trials of the first extinction series. A mean recovery ratio in Table III is the average of the 15 individual recovery ratios in the group.

The mean differences in recovery ratio between any two of the experimental groups are presented in Table IV, together with the probabilities (under 'P') that such differences might have arisen by chance. This table may be compared with Table II in which the reliabilities of the differences between groups were calculated on the basis of mean response amplitudes.

The mean recovery ratios in Table III are means which are based on distributions of ratios. For that reason estimation of the reliability of the differences between any two of these mean ratios in Table IV has been made according to the same *t*-method used for the analysis of the data in Table II. The application of this analysis of the reliability may be limited by the fact that there is, theoretically, no finite upper limit on the ratio scores, and there is, furthermore, no guarantee that the distribution of the means of these ratios will be normal.

In Tables III and IV, as compared with Tables I and II, the effects of the experimental variables are more readily apparent and the differences among groups are more highly reliable. The greater reliability of the differences in Table IV possibly results from the effective manner in

TABLE IV

COMPARISON OF EXPERIMENTAL GROUPS BY PAIRS IN TERMS OF MEAN DIFFERENCES IN RECOVERY RATIO

Experiment I			Experiment II		
Groups	Mean Differences	P	Groups	Mean Differences	P
B–A	.84	<.01	B–A	.27	<.05
B–C	.99	<.005	B–C	.32	<.01
C–A	.15	<.10	C–A	.05	<.40

which the recovery ratio method of analysis discounts much of the variability arising out of individual subject differences in level of responding. This method has the further advantage that it does not give undue weight to those subjects whose responses were consistently of high magnitude. It has the disadvantage that the limitations on accurate reading of the records loom relatively large for the responses of small magnitude. This is possibly significant, inasmuch as the maximal reading accuracy was 0.5 mv., and responses as low as that were sometimes recorded.

Eyeblink.—The interpolated eyeblink procedure was effective in producing conditioned eyeblinks. This is evidenced by the fact that 73 percent of the *Ss*[9] in Groups B and C of Experiments I and II gave three or more conditioned eyeblink responses in the 10 trials of the initial eyeblink conditioning.

The measurement of similarity.—The similarity of the interpolated activities (buzz-eyeblink and light-eyeblink) to the originally extinguished activity (tone-galvanic skin response) may be measured in either of two more or less satisfactory ways. One measure is based on the *a priori* judgment that the one or the other of the two interpolated activities is the more similar to the originally extinguished activity. For the experimental situation presented in this paper, such an evaluation would probably be sufficiently reliable. The interpolated response (eyeblink) was the same in Experiments I and II; only the conditioned stimuli to that response were different. There is every reason to suppose that the 60-cycle buzz stimulus of Experiment I was more similar to the 1000-cycle tone stimulus for the galvanic skin response than was the light stimulus in Experiment II.

A more meaningful and more generally applicable measure of the similarity relationships derives from the extent to which the original galvanic extinction generalized to each of buzz-eyeblink and light-eyeblink (see *The factor of similarity*, above). Unfortunately, the experiment which has been reported here yields only a very insensitive measure of that transfer. It is possible to evaluate the greater or lesser amount of the transfer only by a comparison of the frequencies of eyeblink response during the extinction of the eyeblink in Group C of Experiments I and II. (The eyeblink extinction in these groups follows the galvanic extinction after a five-min. rest.) For Group C of Experiment I the mean number of responses in the first five trials of the eyeblink extinction was 3.30. For Group C of Experiment II the mean number of responses was 3.50. This difference between the mean number of eyeblinks is slight and of no statistical significance. To the extent that the difference has any reliability, however, it would be taken to mean that there was greater transfer of the galvanic extinction to buzz-eyeblink than to light-eyeblink, and that buzz-eyeblink is, therefore, more similar to the extinguished tone-galvanic skin response. It must be noted that the direction of this effect is in agreement with the *a priori* judgment that the interpolated activity of Experiment I (buzz-eyeblink) is more similar to the extinguished tone-galvanic skin response than is the interpolated activity of Experiment II (light-eyeblink).

SUMMARY

It is proposed that spontaneous recovery be regarded as the forgetting (retroactive inhibition) of experimental extinction. Ac-

[9] There were 60 *Ss* in Groups B and C of Experiments I and II. Because of occasional mechanical failure of the eyeblink recording apparatus, eyeblink responses could be measured for only 48 *Ss*.

cording to this view spontaneous recovery is the result of interference with the retention of the extinctive tendency by activity which is interpolated between the last trial of extinction and the test for retention. Retention of the extinctive tendency will be disrupted in the event of the interpolated strengthening of an activity which is similar to the extinguished activity. (A 'similar' activity is one to which the original extinctive tendency generalized.) Retention of the extinctive tendency will be facilitated whenever an interpolated extinction procedure is carried out on an activity which is similar to the originally extinguished activity. By the terms of the analogy of spontaneous recovery with retroactive inhibition, the amount of interference or facilitation of the extinctive tendency will be a function of the similarity between the originally extinguished and interpolated activities.

There is evidence in the literature which permits the tentative conclusion that extinctive tendencies generalize over a wide range of activities. The probability is high, therefore, that a significant number of the secondarily extinguished activities will operate and be strengthened during any normal period of rest. It is suggested that spontaneous recovery occurs because the inevitable interim strengthening of some of these secondarily extinguished activities will interfere with the not-responding tendency which had been set up with reference to the originally extinguished response.

From the assumptions (1) that the capacity for interference is defined by the range of generalization of original responses, and (2) that the extinctive tendency generalizes more extensively than does the corresponding conditioning, it is predicted that the operation of any given interpolated activity will have a greater effect on the retention of the extinction than it will have on the retention of the conditioned response. (For that reason the extinctive tendency will be forgotten more rapidly than the conditioned response in the course of the relatively uncontrolled activity which normally intervenes between learning and test for retention.)

An experiment was designed to provide evidence bearing on the three major aspects of the foregoing hypothesis concerning the nature of spontaneous recovery.

1. *That it is possible to affect spontaneous recovery by interpolating activity in the interval between extinction and test for spontaneous recovery.*—It was found that the interpolation of an eyeblink conditioning procedure increased, over control-rest, the amount of spontaneous recovery of an extinguished galvanic skin response. The interpolated extinction of an eyeblink response decreased the amount of spontaneous recovery as compared with control-rest.

2. *That the degree of the effect on spontaneous recovery varies with*

the similarity between the originally extinguished and interpolated activities.—In one experiment the interpolated activity was the conditioning (or extinction) of an eyeblink for which the conditioned stimulus was a 60-cycle buzz; in the other experiment the interpolated eyeblink was conditioned (or extinguished) to a light. The originally extinguished response was a galvanic skin response which had been conditioned to a 1000-cycle tone. Of the two interpolated activities, the one involving the buzz and eyeblink was judged to be the more similar to tone-galvanic skin response. It was found that the interpolation of the more similar activity (buzz-eyeblink) had the greater effect on the spontaneous recovery of the extinguished galvanic skin response.

3. *That the strengthening of a particular activity has less effect on the retention of a previously conditioned response than it has on the retention of the extinction of that same response.*—The interpolated conditioning of an eyeblink response had an effect on the retention of the extinction of a galvanic skin response. The interpolation of this same conditioning procedure had no measurable effect on the retention of the conditioned galvanic skin response.

(Manuscript received February 25, 1944)

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XUM

PITCH DISCRIMINATION AS A FUNCTION OF TONAL DURATION [1]

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A problem of practical and theoretical importance is the relationship between the duration of a tone and the ease with which its pitch can be distinguished. Two studies may be cited in which this problem has been the primary consideration. The first was that of Anderson (1), who decreased the time of presentation of two successive tones from two sec. to 0.25 sec., and observed a reduction in the accuracy with which his subjects could distinguish a difference of one cycle. More recently, Békésy (2) attacked the same problem, using the duration limits of 0.37 and 0.01 sec., and thus extended the time range to values considerably shorter than any investigated by Anderson. In Békésy's experiment likewise, accuracy of discrimination was found to decrease as the duration of the tones was reduced. The present experiment extends these studies to show more fully the relationship between pitch discrimination and stimulus duration.

Three frequencies, of 128, 1024, and 8192 cycles, were used, and the relative difference limen (i.e., the smallest distinguishable frequency difference divided by the standard frequency) was determined for a series of duration values from the maximum of 0.5 sec. to the shortest period which permitted measurement of the limen. In addition, with a frequency of 1024 cycles, the difference limen (DL) was found for various stimulus intensities (80, 60, 40, 30 and 20 db. above threshold) at two representative duration values (0.1 and 0.35 sec.).

Apparatus

Separate oscillators were used to generate the standard and comparison tones. After being attenuated and filtered, the output of each oscillator was delivered separately to a mechanical timing device, beyond which the two circuits joined and led into a sound-proof room where a magnetic telephone receiver was connected in the common circuit.

The mechanical timer—the Schumann 'Zeitsinn' apparatus—consisted of an arm rotating at a constant speed around a central axis. On the circumference of the circle traced by the arm were clamped two pairs of switches, each pair being part of the circuit of one of the oscillators. On striking the first of each pair of contacts, the revolving arm completed the circuit of the oscillator involved; on striking the second, it broke the circuit. Thus the duration of current flow could be controlled by the speed of rotation of the arm and the distance separating the two switches.

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[1] From the Psychological Laboratory of Princeton University.

Communication with the *S* was effected by a two-way telephone system. In order to eliminate the distraction of sounds from the *S*'s communication receiver, the telephone was cut to a one-way system (from *S* to *E*) while the tones were being presented. As a 'ready' signal, a small light in the *S*'s room could be turned on by the *E*.

Procedure: Experimental Series A

The *S* was seated in the sound-proof room, and the motor regulating the timer was started, a master switch leaving open the circuits of both oscillators. Oscillator 1 was set at one of three frequencies (128, 1024 or 8192 cycles) and oscillator 2 at a frequency above or below the standard by a known number of cycles. The telephone connection from the apparatus room to the sound-proof room was cut and the ready signal given. When the arm of the timer approached the first of the two contacts governing the duration of the standard tone, the circuits of both oscillators were closed at the master switch, and remained closed until both circuits had been completed and broken once by the timer, whereupon the master switch was re-opened.

The calculated durations were verified by an electric timer, which was sufficiently accurate for all but the very short intervals. The latter were checked by means of an oscilloscope, which recorded the number of vibrations present at the membrane of the *S*'s earphone. As the number of vibrations per sec. was known, it was possible to calculate the duration required to give the number of vibrations recorded.

The duration of the standard tone was constant at 0.5 sec., while that of the comparison tone took values from 0.5 sec. to the shortest duration which allowed measurement of the *S*'s DL. The standard always preceded the comparison tone by 0.5 sec. The intensity of all tones was held constant at 60 db. above threshold. The *S* was required to write H or L, according as the comparison tone sounded higher or lower than the standard, with no judgments of 'equal' permitted. He was required further to report when ready for the next pair of tones. While the master switch was open between judgments, the oscillator generating the comparison tone was tuned to the higher or lower frequency in predetermined random order.

Within each set of 100 judgments the frequency difference between the comparison tone and the standard was the same when the comparison tone was higher than the standard as when it was lower than the standard. For example, when the standard was 1024 cycles, there might be 50 comparison tones with a frequency of 924 cycles and 50 with a frequency of 1124. To minimize fatigue, each set of 100 judgments was split into two groups of 50; and each group of 50 contained 25 higher and 25 lower comparison tones, requiring an over-all time of two to three min. There followed a rest period of approximately three min., and then the last 50 judgments of the set were made. For a given frequency of the standard tone and a given duration of the comparison tone, 300 judgments (three sets of 100 each) were made within a period of 30 to 45 min. For the second set of 100 judgments, the frequency difference between the standard and comparison tones was adjusted by an amount calculated (on the basis of results previously obtained in the experiment) to allow approximately 75 percent of correct judgments. A similar adjustment was made for the third set of 100 judgments. The comparison tone was thus presented at six frequencies in all, three higher and three lower than the standard.

From the six accuracy values so obtained, the DL was computed as follows. The three accuracy values in which the comparison tone had a frequency higher than the standard (frequency increment, or Δf, positive) were plotted on a graph having Δf as the abscissa and percent accuracy as the ordinate. A straight line was fitted to the points, and from it could be read the Δf which would theoretically be perceived correctly 75 percent of the time.[2] In the same way, the 75 percent value was found using the three points obtained for Δf negative.

From the interpolation at the 75 percent point for Δf positive and negative two DL's were found, representing respectively the least perceptible rise and the least perceptible drop in pitch. These DL's were averaged to give the figure most representative of the smallest perceptible alteration of pitch under the experimental conditions, and it is this value that will be referred to hereinafter as the DL.

[2] This simple rectilinear interpolation method of estimating the DL seems justified in the light of recent work by Stevens, Morgan and Volkmann (14) and by Flynn (8) on the form of the psychometric function for pitch discrimination. The authors found that a straight line gave a better fit in general than did the traditional normal ogive.

Six *S*s were used in the experiment. A different presentation-order of the three standard frequencies was assigned to each *S*, and for a given *S* all results for each standard frequency were obtained before the next frequency was introduced. The duration values of the comparison tone were randomized. All results obtained in the first experimental session for each *S*, and the first 100 judgments in all later sessions, were discarded. The *S*s were watched closely for signs of fatigue, and most of the experimental sessions were concluded within two hours.

Results: Experimental Series A

Fig. 1 shows the curves obtained for the 128 cycle tone. In this figure, as in those following, the ordinate represents $\frac{\Delta f}{f}$, while time

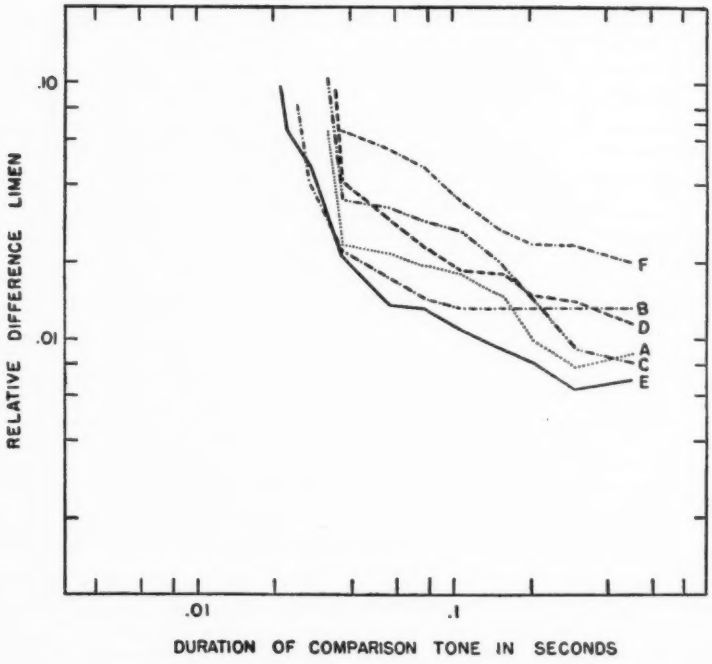

Fig. 1. Increase in the DL with reduction of stimulus duration at 128 cycles. Each curve represents a single subject.

is plotted on the abscissa. It is seen that as tonal duration decreases, $\frac{\Delta f}{f}$ increases. Individual variations are wide, particularly at the short presentation times, although this fact is not immediately apparent on the logarithmic graph. There is evidence that the curve begins to rise more sharply when the very short duration values are

reached; and it will be noted that the largest value found for $\frac{\Delta f}{f}$ was approximately 0.1, beyond which consistent measurements could not be obtained.

Decidedly steeper curves were found for 1024 cycles (Fig. 2) and for 8192 cycles (Fig. 3). At 1024 cycles, the curve does not begin to

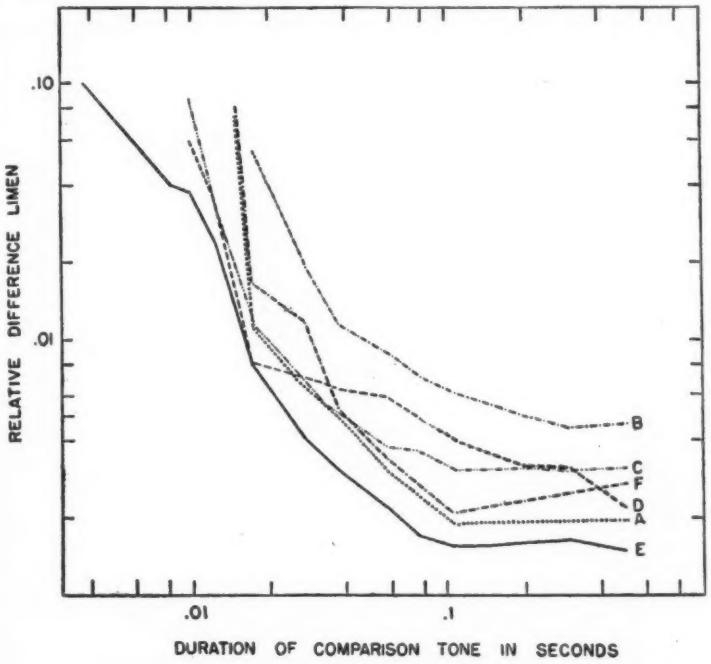

FIG. 2. Increase in the DL with reduction of stimulus duration at 1024 cycles. Each curve represents a single subject.

rise as early as at 128 or 8192 cycles. Comparison of the curves for different frequencies may be made from Fig. 4, which is the linear representation of the averages for the six *S*s at each frequency.

The average values of $\frac{\Delta f}{f}$ on which Fig. 4 is based are given in Table I. Not all the durations listed were used for every *S*. The numbers in parentheses represent the averages in which the values for certain *S*s were found by interpolation.

The results found at the shortest tonal duration which gave a limen for all *S*s (the last possible 'vertical' averages on the graphs)

are given in Table II. The last column represents the minimum number of cycles, obtained as the product of frequency and duration, required for pitch discrimination.

Since the time values in Table II are the smallest which permitted averages of the DL's, they represent the shortest durations which gave a limen for the *S* with poorest pitch discrimination. The

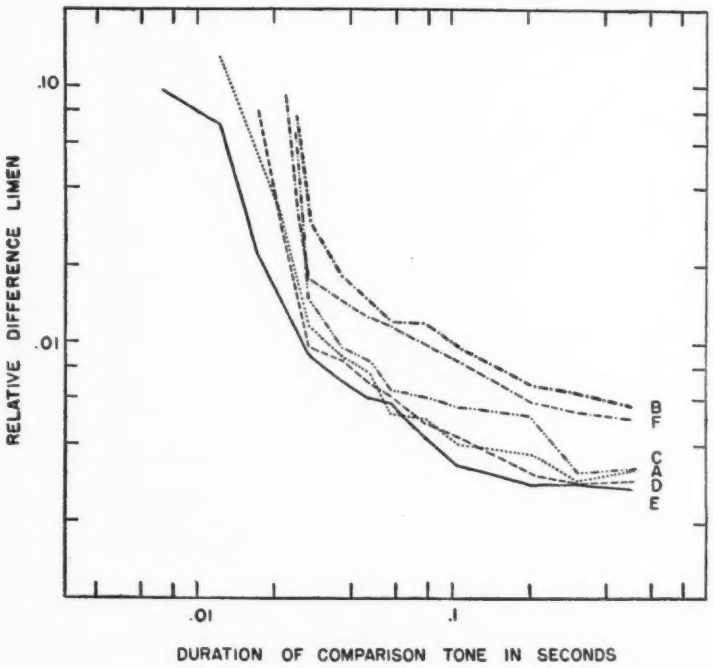

FIG. 3. Increase in the DL with reduction of stimulus duration at 8192 cycles. Each curve represents a single subject.

figures for the average times at which the last measurable DL's for all *S*s were found (the last possible horizontal averages on the graphs) are given in Table III.

In Table IV are given the figures for the shortest durations which gave a limen for the *S* with the best pitch discrimination. Each figure in Table IV refers to the best *S* only.

Comparison of Table IV with Table II shows the wide individual differences in the stimulus duration which is necessary for pitch discrimination.

Discussion

Before comparing the results of the present experiment with those of previous investigations, it is important to consider the physical nature of a brief sound stimulus which is transmitted by a telephone receiver. The diaphragm of the receiver has a certain amount of inertia which must be over come as it is forced into vibration, and so has to build up to the amplitude of oscillation finally reached. If an extremely brief impulse were applied to the membrane, it would do

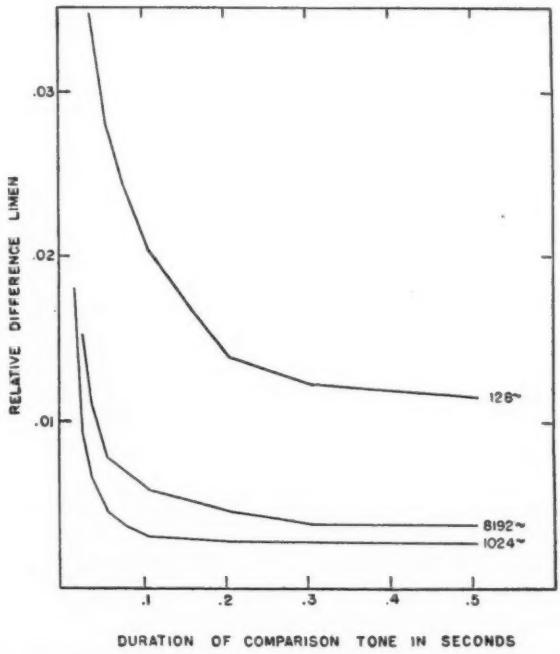

Fig. 4. Increase in the DL with reduction of stimulus duration at 128, 1024 and 8192 cycles. Each curve represents the average of six subjects.

little more than overcome the inertia of the membrane, and would theoretically produce a fraction of a wave representing no one frequency—i.e., having a spectrum composed of all frequencies at equal strength. Hence the pitches of two such impulses could not be distinguished on the basis of vibration rate.

When set in motion, the diaphragm has a 'natural' frequency, at which it would vibrate if no other vibration rate were impressed upon it; but its tendency to oscillate at its natural frequency is

TABLE I

Seconds	DL at 128 cycles	DL at 1024 cycles	DL at 8192 cycles
.5073	.0115	.0028	.0038
.3073	(.0123)	(.0028)	.0039
.2073	(.0139)	(.0028)	.0046
.1573	(.0170)		
.1073	.0205	.0031	.0060
.0773	.0244	(.0038)	.0071
.0573	.0281	.0046	.0079
.0473			.0095
.0373	.0347	.0066	.0112
.0273		.0093	.0154
.0173		.0181	

TABLE II

Frequency (cycles)	Time (sec.)	$\frac{\Delta f}{f}$	Δf	No. cycles ($f \times T$)
128	.0373	.0347	4.44	4.77
1024	.0173	.0181	18.53	17.72
8192	.0273	.0154	126.16	223.64

TABLE III

Frequency (cycles)	Time (sec.)	$\frac{\Delta f}{f}$	Δf	No. cycles ($f \times T$)
128	.0301	.0878	11.24	3.85
1024	.0117	.0760	77.82	11.98
8192	.0181	.0861	705.33	148.28

TABLE IV

Frequency (cycles)	Time (sec.)	$\frac{\Delta f}{f}$	Δf	No. cycles ($f \times T$)
128	.0213	.0965	12.35	2.73
1024	.0038	.0985	100.86	3.89
8192	.0073	.0965	790.53	59.80

opposed by the electrical impulses which are acting upon it at a different rate. During the first vibrations, both the natural frequency and the impressed frequency may be present at the same time.

These early vibrations are known as 'transients.' In their spectrum, the total energy is not narrowly concentrated at the principal vibration rates, but rather is distributed over a wide spread of frequencies. The pitch of the resulting sound is correspondingly indefinite, and the transients are heard as a click.

As the flow of current in the system continues, the vibration rate of the membrane approaches the frequency of the impulses which

sustain its movement, and the transients die out. In the tonal spectrum, a larger percentage of the total energy is concentrated near the impressed frequency, and consequently it becomes easier to assign a pitch to the tone produced (**5, 6**).

The number of transients depends on the 'damping' of the transmitting system—i.e., its resistance to free (or natural) vibration. High damping reduces the relative duration of the natural frequency, and so cuts down the number of transients.

When the current is withdrawn from the receiver, the membrane tends to return to its natural vibration rate. Thus, the shutting-off of the current is followed by another succession of transients, whose number is determined by the degree to which the system is damped.

With the aid of an oscilloscope, the form of the waves produced by the membrane was studied, and the number of transients determined. Approximately the first three waves at 128 and 1024 cycles were transients, and about the first five at 8192 cycles. It will be noted (*cf.* Table IV) that one *S* was able to differentiate the pitch at 128 cycles when only 2.7 cycles were given altogether. As these would presumably all be transients, it is evident that there was a characteristic difference between the transients produced by the two frequencies distinguished.

Kucharski (**9, 10**) has found pitch discrimination possible at much shorter stimulus durations than was the case in the present experiment. In his 1923 paper, he reported a study in which two tones, of 750 and 1000 cycles, were presented successively for a period (0.00054 sec.) sufficient for the transmission of approximately 1/2 cycle of the 1000 cycle wave, and 2/5 cycles of the 750 cycle wave. His seven *S*s were able to distinguish the two pitches without fail, and the more musical of them recognized the interval as a musical fourth. In his later study (1930), Kucharski worked with tones of 100 and 150 cycles, presenting only a single cycle of each. All five of his *S*s were able to distinguish the difference in pitch, and four of them recognized the interval as a musical fifth. Kucharski stated, moreover, that the sound produced by 1/2 vibration at 100 cycles was indistinguishable from that produced by a complete vibration. In this later experimental report, Kucharski included oscillograms of single vibrations obtained from his telephone receiver at the frequency of 200 cycles, and of half vibrations at 100 cycles, noting that sufficient damping is obtainable only if 'relatively weak' intensities of the stimuli are used.

It should be pointed out that Kucharski's 1930 experiment does not constitute a verification of his earlier work, as the frequencies of 750 and 1000 cycles are in the region of the natural frequency of most receivers, so that damping is much more difficult to achieve

near these vibration rates. Moreover, Kucharski gave no indication of the intensities at which his experiments were conducted.

In Kucharski's work, the object was to discover the smallest number of vibrations which would allow the *Ss* to discriminate between the pitches of a pair of tones. For over a century, experiments have been performed on the related problem of the minimum number of vibrations needed to produce a sound judged by the subject to have 'pitch' or 'tonality' (**3, 4, 9, 13**). The private and undefined nature of the criterion and the faulty control of stimuli in these 'subjective' studies render the wide differences in the conclusions understandable, and make absolute comparison of results dangerous. It is of interest, however, to compare the general range of the results obtained in these studies with the minimum number of vibrations needed to give a DL in the present experiment. To produce a sensation meeting the criterion of 'tonality,' most workers have found from two to 15 cycles necessary in the low and middle frequency ranges; while in the present experiment it was found that the number of vibrations must be of the same general magnitude (from 2.7 to 17.7 cycles) to allow objective measurement of pitch discrimination.

In only one of the early 'subjective' studies was pitch perception obtained at durations comparable to those at which Kucharski obtained discrimination. Cross and Maltby (1891) used a telephone receiver to pick up the sound from an electrically driven tuning fork. They found that a tone of 256 cycles could be recognized from 70 to 80 percent of the time if presented for a period which theoretically would allow only .88 of the vibration to be completed; while a tone of 512 cycles required 1.78 cycles for its recognition. It is entirely probable, however, that the damping of their receiver was insufficient to eliminate all transients, and therefore that a greater number of vibrations were present than the experimenters believed.

Perhaps the best available 'subjective' investigation has been that of Buerck, Kotowski and Lichte (**4**), who have undertaken several studies related to the problem at hand. The average stimulus durations required for their *Ss* to identify the pitches of tones at different frequencies may be read from the graph presented by the authors in a 1935 paper. For the three frequencies used in the present experiment, the figures of Buerck, Kotowski and Lichte are the following:

Frequency (cycles)	Minimum time required (sec.)	No. cycles ($f \times T$)
128	.030	3.84
1024	.012	12.3
8192	.020	164.

The close correspondence of these figures with the smallest average

number of vibrations which in the present study produced a measurable DL (*cf.* Table III) is noteworthy.

In the experiments so far reviewed, the object was to study pitch perception at the shortest possible stimulus duration. However, in the present experiment, and in the investigations of Anderson (1) and Békésy (2) mentioned in the introduction, the purpose was to vary stimulus duration through several values, and to measure pitch discrimination at each duration value.

The ability of Anderson's four *S*s to differentiate between two successive tones which differed by one cycle varied with duration as follows:

Duration (sec.):	2	1	0.5	0.376	0.25
Percent accuracy of perception:	88.3	80.4	77.5	76.0	68.4

As Anderson did not report the frequency of the standard tone used, it is difficult to compare these figures with the data herein reported.

Békésy's results were obtained from two *S*s, with a standard tone of 800 cycles. As his measure of discrimination he employed the relative DL. With intensity held constant at 40 db. above threshold, Békésy's values of $\frac{\Delta f}{f}$ are the following:

Duration (sec.):	3.7	2.45	1.2	.051	.02	.01
Subject A: $\frac{\Delta f}{f}$:	.0024	.0026	.0034	.0056	.0068	
Subject B: $\frac{\Delta f}{f}$:	.0032	.0034	.0040	.0048	.0070	.0097

Interpolation on the graph of the results obtained in the present experiment with the 1024-cycle tone (Fig. 4) gives the following values of $\frac{\Delta f}{f}$:

Duration (sec.):	3.7	2.45	1.2	.051	.02
Average: $\frac{\Delta f}{f}$:	.0028	.0028	.0031	.0046	.0155

It is seen that for long stimulus durations, Békésy's results are very similar to those found in the present experiment. For durations below .05 sec., however, the DL's found in the present experiment are considerably greater than those reported by Békésy. An example of the magnitude of this difference may be seen by comparing the values at .02 sec. in the above tables.

Experimental Series B

In the preceding discussion, accuracy of pitch discrimination has been treated as a function of duration only. Among sensory phenomena, however, there are many instances in which the two variables

of duration and intensity are so related that a decrease in either variable may be compensated for by an increase in the other. A light stimulus, for example, remains equally visible as its duration is decreased, provided its intensity is suitably increased at the same time. When the light is near threshold value, the relationship of intensity and time may be expressed by the formula $I \times T = K$, where K represents the visibility of the stimulus.

Lifshitz (**11**, **12**) has suggested that in audition a reciprocal relationship exists between duration and the logarithm of intensity (rather than between duration and intensity, as is the case in vision). This author has stated that for short tones between 34 and 84 db. above the reference intensity of 10^{-16} watts/cm.^{2}, the relationship $L_0 \times T = K$ holds true. L_0 here stands for number of decibels, or $10 \log \frac{\text{stimulus intensity}}{\text{reference intensity}}$.[3]

The use of logarithmic 'units' (decibels) to represent intensity in such an equation introduces a difficulty because of the fact that the ratio of two intensities which are expressed in db. above a given reference intensity is altered if the reference intensity is altered. The specific nature of the difficulty thus introduced may best be understood from an example. If a stimulus were 20 db. above a given reference intensity, and T were .04 sec., then K (their product) would equal 0.8. If the number of decibels were doubled, the formula would require that T be reduced to .02 sec. to keep the product constant at 0.8, and thus to compensate for the intensity increase. If, however, a different reference intensity be selected 10 db. below the first, the stimuli which were 20 and 40 db. above the original reference level will be 30 and 50 db., respectively, above the new reference intensity. Now, if we multiply number of decibels by time, we find that the products are $30 \times .04 = 1.2$ and $50 \times .02 = 1.0$. In other words, when decibels are expressed in terms of a new reference level, a different time reduction is required to balance the equation. It is obvious, however, that the time reduction required to keep discrimination constant is the same as before, since we have changed nothing but the way in which the original physical quantities are expressed. It is apparent, then, that the formula can be valid only when the number of decibels is expressed in terms of some one reference intensity. If any other reference intensity is used, a new formula will be required to fit the data.

Lifshitz (**11**, **12**) has reported two studies in which he has tested the applicability of the formula, but he has not used the same refer-

[3] When written as $I_0 \times T = K$, the equation is misleadingly suggestive of a function in which all units are linear. It is more appropriately written $T \log I_1/I_0 = K$, where I_1 = stimulus intensity and I_0 = reference intensity.

ence intensity in both studies. The difference (of four db.) between his two reference points is insufficient to affect the figures of either study seriously; but it must be emphasized that as it is impossible for more than one reference intensity to be appropriate, the appropriate one must be specified if the formula is to be of value. Hence, the figures of one or the other of Lifshitz's studies stand in need of revision, if they are to represent confirmation of the equation proposed.

The difficulty of the dependence of the equation's validity on the choice of reference intensity may be overcome by the introduction of an added constant into the formula as a multiplier of the energy ratio: i.e., $T \log C \frac{I_1}{I_0} = K$. The constant C may be assigned different values to compensate for changes in the size of I_0. The fact that no such constant was required by Lifshitz to fit his data means that C has approximately unit value at the intensities which he used.

If a reciprocal relationship between duration and some function of intensity holds true in audition, as Lifshitz suggests, the DL's obtained by a reduction of stimulus duration in experimental series A of the present experiment could likewise be obtained by a suitable intensity decrease. In order to investigate this possibility, a further series of experiments (experimental series B) was undertaken, in which stimulus intensity was varied through a wide range. Three *S*s (C, D and F of series A) were used, with a standard tone of 1024 cycles. At the two durations of 0.1 and 0.35 sec., each *S*'s DL was found at the intensities of 80, 60, 40, 30 and 20 db. above threshold. The presentation order of these intensities was randomized. In all other particulars, the procedure for series B was the same as that for series A.

The values of the DL obtained in series B are seen in Table V.

TABLE V

Decibels	DL at .035 sec.	DL at .10 sec.
80	.0064	.0032
60	.0059	.0032
40	.0079	.0043
30	.0098	.0053
20	.0122	.0063

It is observed that, in general, discrimination was better at strong intensities of the stimuli. There is one exception, however. A tone of 80 db. sounded for .035 sec. gave a larger DL than did a similar tone of 60 db. Further investigation with greater stimulus intensities would be necessary to determine whether or not this reversal of the trend is significant.

Of particular note is the fact that a decrease of intensity at .035 sec. had a markedly greater effect on the DL than did a similar decrease at .1 sec.

The results of the present experiment show that an intensity reduction raises the DL for pitch, as does a duration reduction. By comparing the results obtained in series B by varying intensity with those found in series A by varying duration, it is possible to test the strength-duration formula proposed by Lifshitz. In calculating the intensities of the present experiment, the threshold of audibility (6.5×10^{-16} watts/cm.^{2}; see Fletcher, **7**) was used as the reference level. Thus, in testing the applicability of the formula $T \log C \frac{I_1}{I_0} = K$ in the present experiment, the constant C must be given the value of 6.5 to reduce the reference intensity to that used by Lifshitz (10^{-16} watts/cm.^{2}). The introduction of a C of 6.5 is equivalent to the addition of approximately 8 db. to the number of decibels used to express each stimulus intensity, since $10 \log 6.5 = 8.1$.

In terms of the formula, if for any two tones the product of decibels and duration is the same, their DL's will be the same. To test the validity of the formula, we have simply to find from series A the tones for which the product of duration and intensity equals the product of a different duration and intensity used in series B, and compare the DL's of the tones thus matched.

Tables VI and VII show the DL's of tones in the two experimental series which have equal strength-duration products (K).

TABLE VI

Decibels	Seconds	K ($db \times T$)	DL at .035 sec.	Seconds	K ($db \times T$)	DL at .1 sec.
88	.035	3.08	.0064	.100	.88	.0032
68	.035	2.38	.0059	.100	.68	.0032
48	.035	1.68	.0079	.100	.48	.0043
38	.035	1.33	.0098	.100	.38	.0053
28	.035	.98	.0122	.100	.28	.0063

TABLE VII

Decibels	Seconds	K ($db \times T$)	DL	Seconds	K ($db \times T$)	DL
68	.0453	3.08	.0050	.1294	.88	.0030
68	.0350	2.38	.0060	.1000	.68	.0032
68	.0247	1.68	.0093	.0706	.48	.0039
68	.0196	1.33	.0109	.0559	.38	.0044
68	.0144	.98	.0460	.0412	.28	.0052

Table VI is a re-statement of Table V in series B, except that in Table VI, 8 db. has been added to each value in the first column of Table V, so that decibels are now expressed with reference to 10^{-16} watts/cm.^{2}; and the values of K (number of decibels multiplied by duration) have been included.

In experimental series A, all stimuli were 68 db. above 10^{-16} watts/cm.^{2} (i.e., 60 db. above threshold). In Table VII are shown the durations which, when multiplied by 68, give a product K equal to each K shown in Table VI. For example, in Table VI it is seen that when an 88 db. tone is presented for .035 sec., $K = 3.08$; and in Table VII it is found that a 68 db. tone sounding for .0453 sec. also gives a K of 3.08. The DL for the former, however, is .0063, while the DL for the latter is .005. While some of the DL's in Table VI do not differ greatly from the corresponding DL's in Table VII, wide differences are evident in many cases. Since the DL's of the two tables would be identical if the formula were correct, it is apparent that the data of the present experiment do not support the equation $T \log C \frac{I_1}{I_0} = K$ as a formula expressing the relationship between time and intensity in audition.

Summary

The results obtained in the present experiment on the relation of stimulus duration to pitch discrimination may be described as follows. As the duration of tonal stimuli decreases, accuracy of pitch discrimination likewise decreases. For a tone of 1024 cycles at 60 db. above threshold, the effect of stimulus duration on pitch discrimination is slight until the duration is reduced to 0.1 sec. Thereafter, accuracy of discrimination declines rapidly, until it is reduced essentially to zero around 0.01 sec. For a tone of 8192 cycles at 60 db. above threshold, pitch discrimination is noticeably blunted when one of the tones to be compared has a duration of less than 0.25 sec.; and accuracy reaches a virtual zero when duration is reduced to approximately 0.02 sec. For a tone of 128 cycles presented at 60 db. above threshold, accuracy of pitch discrimination decreases if the duration of one of the stimuli is reduced to less than 0.5 sec. The curve representing decrease in accuracy is thereafter less steep, as duration is reduced, than is true at 1024 or 8192 cycles; but the virtual zero point of discrimination accuracy is reached sooner, at about 0.03 sec.

A decrease in stimulus intensity likewise produces a reduction in accuracy of pitch discrimination. This reduction is greater at relatively short durations. The data do not support the hypothesis that discriminability varies as the product of intensity in decibels and duration in seconds.[4]

(Manuscript received March 22, 1944)

[4] The guidance of Dr. E. G. Wever and Dr. H. S. Langfeld in the conduct of this experiment is gratefully acknowledged.

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STUDIES IN COLOR BLINDNESS: I. NEGATIVE AFTER-IMAGES *

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INTRODUCTION

Negative after-images "have been used from the first as supplementary evidence of dichromatic vision" (3, p. 328). Further investigation is indicated, however, by the controversial results of prior studies.

In 1925 Collins (1) had nine color-blind *Ss* name the colors of their negative after-images to six hues of Bradley paper. All *Ss* reported blue to yellow and orange, and reported yellow to blue and violet. To red, seven reported white or gray, one reported green, and one pale green. To green, five reported white or gray, two pink, 1 red, and one blue.

Murray (3, p. 328) reports that negative after-images to blue and violet or red and yellow exposed side by side and projected on a gray ground are indistinguishable by the dichromat, being "yellowish in the first case" and "blue in the second."

Wiltberger's test (4) for color-blindness assumes that individuals who are actually red-green blind obtain white after-images to his red and blue-green chips of "high chroma, high value and of accurate hue," while those who are only color-weak report after-images of the complementary hue.

SUBJECTS

The experimental group consisted of 20 individuals reporting not more than two 'normal' responses on the 5th edition of the Ishihara test for color-blindness. The two dichotomous plates definitely classified 10 of the above as deuteranopes and seven as protanopes, while the other three were 'ambiguous,' since both the red and red-purple digits of the double numbers were invisible to them. Eight persons giving all 'normal' responses were used as controls.

The Ishihara test has been accused of "throwing down color-weak along with color-blind" (3, p. 330), and after completion of the experiment a further classification of the experimental group, into the above categories, was found possible on the basis of their after-images. Twelve of these *Ss* were classified as color-blind, since they showed no differential after-images to red, yellow, and green or to blue and purple. The other eight *Ss* were classified as color-weak, since their after-images approached those of the normal controls. Their original classification by the Ishihara test was:

* This first study was completed as an M.A. thesis under the direction of Dr. J. F. Dashiell. Dr. R. J. Wherry supervised the statistical analysis and aided in the interpretation.

	Color-Blind	Color-Weak
Protanope	5	2
Deuteranope	5	5
Ambiguous	2	1

The justification for this classification will be found in the results to follow.

Procedure

The stimuli were 1½ in. squares of Munsell paper (2) with a value (brightness) of 5 and a chroma (saturation) of 6 on the Munsell scale. The hues were red, yellow, green, blue, and purple. Illumination by three 60-watt daylight bulbs was kept constant. Each stimulus square was presented in the usual manner, and the after-image was then projected on a gray background. Fixation periods were of 30 to 40 sec. with rest periods of three to five min. duration. Each *S* had four experimental sessions at least 24 hours apart, during each of which the five hues were presented. The *S*'s task was to find the color chip in the Munsell book whose hue, saturation, and brightness most nearly approximated his after-image. To facilitate this, as well as to prevent the use of color names, the letters in the Munsell book were converted into numbers. The numbers on the circle of colors in the front of the book corresponded to the page numbers containing the various saturations and brightnesses for each hue. Thus, the *S* picked from the circle the color nearest to his after-image, turned to the corresponding page, and matched his after-image there as accurately as possible. Each *S* was urged to consult adjacent pages in the book and to repeat the after-image as many times as necessary to be sure of his choice.

Frequency distributions were made by using the arbitrarily assigned page numbers of the Munsell book as hue values. Since each page represented an equal distance on the Munsell Hue Circle, it was deemed justifiable to convert these to prime numbers. This scale is, of course, circular rather than linear, so that 1 = 21, 2 = 22, etc.; and such substitutions were employed when distributions were in the vicinity of PR and R. Similar distributions were made from the already present Munsell numbers for brightness and saturation. From these numbers means and sigmas were calculated. Since no number in the sequence could be assigned logically to gray, gray after-images were not included in any of the distributions. One protanope reported gray after-images four times out of the four exposures to the red stimulus. Each of two deuteranopes reported gray to the green stimulus three times out of four. No other reports of gray occurred more than once per *S* per color.

Results

Critical ratios, based on means and sigmas for the *hues*, indicate that the classification made above was sound, since those classified as color-weak did fall between the color-blind and the normal, being unreliably distinguished from the latter in three instances (to Y, G, and B stimuli), and from the color-blind to the Y stimulus. The truly color-blind and the normals were reliably different for all hues. The actual critical ratios follow:

Stimulus Hue	CB vs. Normal	CW vs. Normal	CB vs. CW
R	11.30	4.11	8.71
Y	4.67	2.70	1.45
G	8.38	1.92	5.93
B	7.97	0.22	8.32
P	9.95	3.90	4.23

The mean hues (reconverted to color names) and the numerical sigmas for these three groups were:

Stimulus Hue	Color-Blind		Color-Weak		Normals	
	M	σ	M	σ	M	σ
R	PBP	1.52	B	.95	BGB	1.29
Y	PBP	1.38	BP	1.35	BPB	1.03
G	PBP	1.74	PRP	1.72	RP	1.81
B	Y	1.05	YR	1.69	YR	1.93
P	YGY	.92	YG	1.24	GYG	.98

The above means of the hues are plotted in Fig. 1 to give a visual presentation of the results.

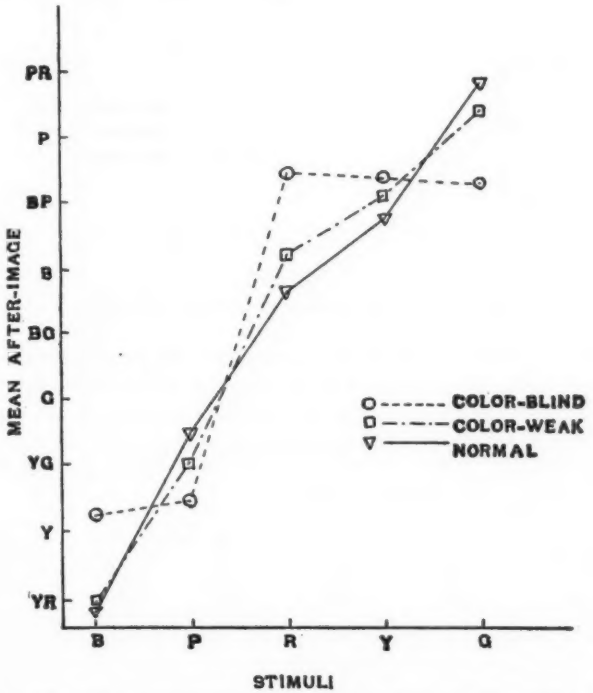

FIG. 1. Mean hue of negative after-images for color-blind, color-weak, and normal *Ss*.

Critical ratios based on the means and sigmas for brightness and saturation also disclosed the value of the classification color-blind vs. color-weak. The means for the *brightness* of the after-images reported by the three groups appear in Fig. 2. Visual inspection reveals that the normals and color-blind lie close together, while the color-weak consistently report after-images of lower brightness.

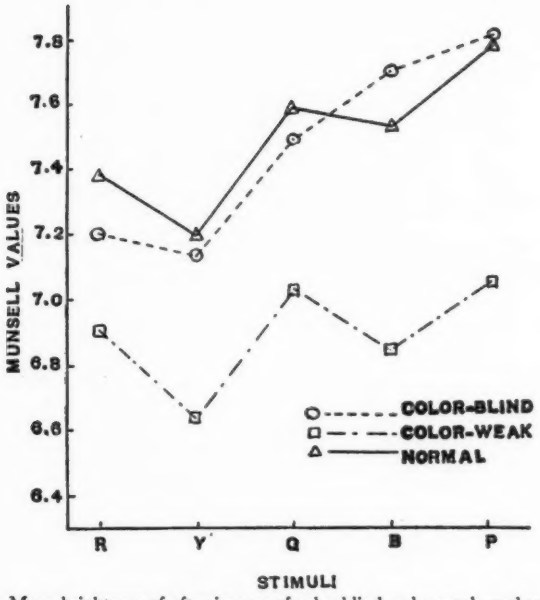

FIG. 2. Mean brightness of after-images of color-blind, color-weak, and normal *Ss*.

There also appears a trend for reports of higher brightness at the blue-purple than at the red-yellow end for the two former groups, while no such trend is apparent for the color-weak.

The validity of the differentiations observed is shown by the following critical ratios. The critical ratios between the means of the three groups for each hue were:

	R	Y	G	B	P
CB–CW	1.42	3.16	3.48	7.75	7.14
CW–N	3.53	4.18	4.24	5.48	6.32
CB–N	0.85	0.47	0.77	1.55	0.32

It can be seen that with one exception, all the critical ratios on the first two lines are reliable while none on the bottom line are reliable.

The higher brightness reports for the blue-purple as contrasted with the red-yellow for the color-blind and normals, but not for the color-weak, is substantiated by the following critical ratios:

		R	Y
P	CB	*3.10*	*5.53*
	N	*3.10*	*5.90*
	CW	1.23	2.97
B	CB	*2.50*	*4.49*
	N	*1.15*	*3.04*
	CW	0.46	1.44

With respect to the reported *saturation* of the after-images the color-weak are again separated from the normals, while the color-blind parallel first one group and then the other, due to a greater demarcation of a trend toward higher saturations at the blue-purple rather than the red-yellow stimuli present in all groups to some degree. These characteristics of the three groups are shown by the means in Fig. 3.

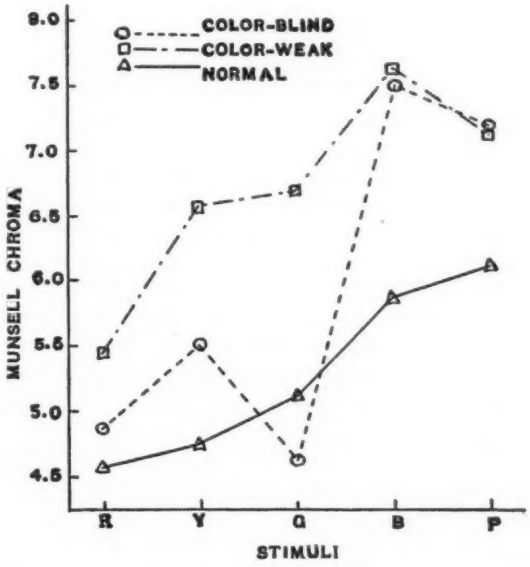

FIG. 3. Mean saturation of after-images for color-blind, color-weak, and normal *Ss*.

The validity of these differences is shown by the critical ratios of differences in the saturation means for the three groups:

	R	Y	G	B	P
CB–CW	*1.75*	*2.80*	*5.94*	0.45	0.17
CW–N	*2.53*	*4.97*	*3.43*	*4.10*	*2.36*
CB–N	0.83	2.07	1.05	*4.00*	*2.92*

The trend toward increased saturation of after-images at blue-purple as contrasted with the red-yellow end and sharper break in the case of the color-blind is shown by the following intra-group critical ratios between hues.

		R	Y	G	B
P	CB	*7.44*	*5.11*	*7.80*	1.08
	N	*3.86*	*3.52*	2.00	0.54
	CW	*4.76*	*1.44*	1.19	1.32
B	CB	*8.43*	*6.03*	*8.75*	
	N	*2.98*	*2.63*	1.42	
	CW	*6.95*	*2.96*	2.84	
G	CB	0.64	2.33		
	N	1.19	0.82		
	CW	4.12	0.37		
Y	CB	1.71			
	N	0.52			
	CW	3.35			

Discussion and Conclusions

1. The failure of the Ishihara test to differentiate anomalous trichromats from dichromats (both of red-green variety) necessitated a reclassification of the 20 *S*s into color-weak and color-blind. This reclassification on the basis of the hues of the negative after-images reported during the experiment proved worthwhile, as shown in Fig. 1. After the *S*s had thus been classified by *hues* of after-images, the data on brightness and saturation for the two groups further substantiated this division (see Figs. 2 and 3). The fact that 'color-blind' persons, as selected by certain tests, may be subdivided into color-blind and color-weak offers a partial explanation of the ambiguity of Collins' results, particularly the normal and weak normal responses to red and green.

2. The following characteristics of the truly *color-blind* with respect to negative after-images were revealed by inter- and intra-group comparisons:

A. Hues:

a. The mean hues of after-images for the color-blind are restricted to yellows and blues, the former being reported to B and P and the latter to R, Y, and G. This substantiates Murray's reports of 'blue' and 'yellowish' for dichromats with the double presentation, although only one report of blue (to G) was obtained by Collins with R and G stimuli. This attenuated distribution agrees with the reports of a few monocularly red-green blind individuals that the spectrum is reduced to yellows and blues (**5**).

b. The only exception to the yellow-blue restriction was the report of gray when the stimulus or after-image fell in the neutral zone. This showed a difference for protanopes and deuteranopes, since one protanope consistently reported gray to *red*, while two deuteranopes reported gray three times out of four to *green*. This finding would logically point to a variability in the neutral zone. The variability would explain the remaining ambiguity in the Collins

findings, and also indicates that while the Wiltberger test would not 'throw down' the color-weak as does the Ishihara, it would not reliably catch all the color-blind.

B. Brightness:

The brightness of the after-images for the color-blind was not reliably different from the normal to any stimulus, although reliably different from the color weak.

C. Saturation:

With respect to saturation, the color-blind were unreliably differentiated from the normals for reports to R, Y, and G stimuli, although reliably different from the color-weak. On the other hand, they were reliably different from the normals for reports to B and P, here being unreliably differentiated from the color-weak. The increase in reported saturation at the blue-purple end as against the red-yellow end is much more marked for the color-blind than for the color-weak and normals.

3. The *color-weak* are shown to be distinguishable from the normal as are the color-blind but not on the same basis. The following characteristics of their negative after-images were revealed:

A. Hues:

On the whole, the color-weak report hues paralleling the normal *Ss*, being for the most part unreliably different from theirs. In so far as deviations do occur, these lie between the normals and color-blind, but much closer to the normals.

B. Brightness:

The color-weak are reliably differentiated from both the normals and color-blind, in that they report much lower brightness for after-images to all hues, although at this lower brightness level their graph approximates the normal slope.

C. Saturation:

With respect to saturation, the color-weak again roughly parallel the slope of the normals but at a reliably higher level.

4. Findings on the after-images of the *normal Ss* were as follows:

A. Hues:

As expected, the after-images of the normals were the complementary hues of the stimuli.

B and *C.* Brightness and Saturation:

One might have expected uniform reports of brightness and saturation for all hues, since the Munsell stimulus papers were of the

same 'value' and 'chroma.' Actually, the normals reported generally reliably higher saturations and brightnesses at the blue-purple than at the red-yellow end. While this rise was steady for saturation, there were slight downward displacements for Y and B on brightness, which also occurred for the color-weak but not at B for the color-blind.

Summary

Twenty Ss originally classified as color-blind by the Ishihara test were reclassified as color-weak (eight) and color-blind (12) on the basis of reliable differences between the hues of their negative after-images matched against Munsell color chips. Further substantiation of the validity of this division was obtained by comparing the normals, color-weak, and color-blind on saturation and brightness of after-images. Each of the three groups was found to be reliably differentiated from the other two on the basis of one or more color qualities, as indicated below:

1. With respect to *hue* the normals showed the expected complementary responses, whereas the color-blind were restricted to reports of blue or yellow for all hues, except for a few reports of gray when the stimulus or after-image fell in the neutral zone. The color-weak, on the other hand, were much like the normals with respect to hue, but were in all cases removed a usually unreliable amount in the same direction as the color-blind.

2. With respect to *brightness* the color-blind selected chips unreliably removed from those chosen by the normals, whereas the selection of the color-weak were always reliably less bright. A tendency for lowered reports to blue and yellow stimuli common to both the normal and color-weak was absent for the color-blind to the blue stimulus.

3. With respect to *saturation*, the color-weak were again reliably differentiated from the normals by their selection of more saturated chips. The color-blind selected low saturations at the 'yellow' (R–Y–G) portion of the color circle, as did the normals, whereas at the 'blue' (B–P) portion they chose high saturations as did the color-weak.

(Manuscript received March 15, 1944)

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FACTORS IN MASSED AND DISTRIBUTED PRACTICE

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In an earlier paper I concluded from an analysis of the data on massing and distribution of practice that there was experimental evidence for a dual division of learning into 'achievement of insight' on the one hand and 'fixation' on the other. I also predicted that if (and as long as) a novel configuration were unstable, massing of repetitions would be more economical than distribution, since if the configuration were once lost the preliminary process of problem-solving must be gone through again. On the other hand, the fixation process was said to be favored by distributed practice. Two experiments with puzzles were reported supporting this prediction, showing massed practice more economical in early, and distribution in later, trials, with distribution having still more advantage when the task was relearned after a delay of several weeks (**1**).

Since that paper was published, however, I have found reason to believe that (*a*) the primary factor in the advantage of massing is not achievement of insight as such, but the instability of the configuration. In other words, those materials which show a sharp drop in the forgetting curve should profit by massing of repetitions, particularly in the early trials. Now, a rapid drop near the beginning of the forgetting curve is not peculiar to puzzle-solving, nor indeed would this be selected by most observers as the most typical example. Far more familiar is the rapid forgetting of nonsense materials. It seemed probable, therefore, that nonsense materials would also show advantage for massed practice, provided the time interval between trials in the distributed procedure were considerably greater than the two-minute period shown by Ward (**8**) to yield reminiscence rather than forgetting.[1]

In addition, it appeared plausible that two secondary factors might also favor massed procedure. (*b*) The first of these is freedom from interfering impulses set up within the learning process. It

[1] It should perhaps be noted that on this point there is no conflict between Hovland's and my assumptions (5). Rather our results lie in the same continuum. Hovland, deducing from Ward's findings (8) that the forgetting curve for nonsense materials first rises and then falls, further postulated and proved that with nonsense syllables a six-second interval is less economical than a two-minute interval. I merely carry Hovland's logic a step further and argue that a somewhat longer interval between trials (as 24 hours), since it strikes the forgetting curve at a low point, should be less economical than a short (30-second) interval.

should be evident that the hypothesis stated in the previous section implies that any advantage of massing is primarily due to later trials succeeding earlier at an interval so short that the positive effects of learning (those which facilitate improved performance) are not dissipated. But if, as is generally accepted, performance in a learning situation is a resultant of positive and negative influences, the advantage of massing should be more evident to the degree that negative effects are excluded. It should be noted that this would be even more the case if negative effects are forgotten more quickly than positive, for in that event negative effects would accumulate more rapidly in massed than in distributed practice. However, such an assumption is not necessary to my theory, since in the absence of negative effects any decrement in the traces of positive learning during the interval between trials would favor massed practice.

Now, it is reasonable to suppose that negative or interference effects are at a minimum with materials near the memory span in amount, since in such cases performance may be errorless on the second (massed) trial. Moreover, since Hovland has shown that for nonsense materials interference increases with length of list (**6**), it would seem that some light might be thrown on the role of interference in massing and distribution by varying the amount of material. This was done in another study by the writer (**2**), which showed that by the error criterion massed practice (5–10 seconds between trials) has a marked advantage over distributed practice (24-hour interval between trials) in early trials with 4-unit Warden U-mazes, but that this advantage was smaller with 8-unit and still less with 12-unit mazes.

(*c*) The other secondary factor that I suspect favors massing, is the degree of difficulty that confronts the *S* as a consequence of forgetting. In my work with puzzles it is probable that this was an important factor of advantage for massing, since in early trials the configuration was unstable and the puzzle once forgotten was difficult of re-solution. But in memorizing nonsense materials by the usual techniques the penalty of forgetting is small, since if an *S* cannot reproduce an item he proceeds almost immediately to the next item. In U-type mazes, also (with returns prevented), a wrong choice means only one error and a few seconds delay. Accordingly, in the experiments to be reported I attempted to increase the advantage of massing by increasing the number of culs-de-sac at each choice point.

The basic assumption here is of course my previous one that the kind of learning favored by massed practice is 'memory' of the correct solution. Then if *S* forgets what he did at a given choice point on a previous trial, he must explore a large number of alternatives before again finding the true path.

Experiment I

The first experiment was conducted at Acadia University in the winter of 1940–41. The apparatus was a multiple unit spider maze previously built for work on the whole-part problem (3). The spider maze is an elevated finger maze with eight paths on each unit—entrance, exit, and six blind alleys. For the distribution of practice experiment one change was made. On each unit a nail was driven between two of the staples, and the units of any given maze pattern were so connected that the entrance to each unit was immediately adjacent to one such barrier. Thus, formerly the *S*s had to learn not only whether to go to the right or the left, but also which path was the true exit. Now they had only to learn the latter, since the barrier just at the right or left of the entrance permitted them to go round the central block in one direction only. The mazes were nonsense in character. That is, there were no serial relations between successive numbers, and familiar sequences were avoided.

Two *S*s, (C) and (W), learned in order four 8-unit, four 16-unit, and four 8-unit maze patterns. Subject C learned these patterns by massed or distributed procedure in the order DMMDDMM-DDMMD; subject W, in the order MDDMMDDMMDDM. With massed practice 20 sec., with distributed practice one day, elapsed between trials. The interval between one maze and the next was never less than two days. Each maze pattern was learned to the criterion of one errorless trial. Subject C was already familiar with the learning task, having served as *S* in an intensive series of experiments with the spider maze some two years previously. Subject W had not before confronted spider maze problems, but had some experience with other mazes. Both *S*s were given considerable preliminary practice with spider maze patterns, and were taught a method of learning which consisted in 'trying' the paths on each maze unit in order beginning at the point of entrance, numbering these paths from 1 to 8, and learning the sequence of maze units by memorizing the order of numbers corresponding to the true path on successive units. This method was found to be highly efficient by other *S*s, but had not been discovered by subject *C*. Both *S*s were cautioned not to think of the maze patterns between trials, and reported no difficulty in following this instruction. Neither *S* had any significant knowledge of earlier experimental results in massed and distributed learning.

Results

As in other studies with returns prevented, there is no difference between the relative economy of massing and distribution on the first trial. The data for trials other than the first are presented in Table I. To avoid decimals the scores are in totals; averages may be obtained by dividing each number by two.

The data in Table I may be summarized under several headings.

(1) Massed practice is markedly superior to distributed practice throughout. The superiority appears in the data for both *S*s and for all stages of learning. Each group of four mazes with either *S* shows massed practice more efficient by all three measures of learning. The advantage is greatest with errors, next with time, and least with trials. About 2.5 times as many errors were made and 1.8 times as many trials were required in distributed as with massed practice.

(2) There are no reliable differences between the length-difficulty ratios in massed and distributed practice. For errors, difficulty is proportional to the square of the number of units of maze pattern.

(3) Practice effects are marked. The two *S*s learned the first four 8-unit mazes with an average of 50 errors, and the last four 8-unit mazes with an average of 15 errors per maze. However, there are no reliable indications that the relative economy of massing

TABLE I

TOTAL ERRORS, TIME IN SEC., AND TRIALS FOR TWO Ss LEARNING MULTIPLE UNIT SPIDER MAZES BY MASSED AND DISTRIBUTED PRACTICE

Size of maze	Massed			Distributed		
	Errors	Time	Trials	Errors	Time	Trials
	Subject C					
8-unit	20	284	5	109	817	10
16-unit	59	834	8	251	1807	16
8-unit	10	120	3	21	312	6
	Subject W					
8-unit	103	972	11	169	1369	16
16-unit	218	3119	17	492	5587	27
8-unit	28	373	5	57	608	8

and distribution is affected by practice. Massing is superior to distribution equally at the beginning and end of the experiment.

(4) After trial 1 the curves for massed and distributed practice have in general the same negatively accelerated shape. The error curve for massed practice, however: (*a*) from trial 2 on is markedly lower than the corresponding curve for distributed practice; (*b*) shows a much greater drop from trial 1 to trial 2. With massed practice slightly more than one-quarter and in distributed practice about one-half as many errors appear in trial 2 as are found in trial 1; (*c*) meets the baseline after fewer trials than is the case with distributed practice.

DISCUSSION

The spider maze not only shows a much greater advantage for massing than was the case with the U-maze, but this advantage appears in the error, time, and trial measures, and for all stages of the learning process, instead of (as with U-mazes) only for errors in early trials (**2**). What differences in the two experiments might account for this increased superiority of massed procedure?

(*a*) Since both U-mazes and spider mazes were nonsense in character and both learned by finger-tracing, blindfolded, we may assume that forgetting occurs at a comparable rate for both, and it is extremely unlikely that the differences in relative advantage of massing and distribution can be attributed to differences in rate of forgetting.

(*b*) With the U-maze the size of the maze was found to be nega-

tively related to the superiority of massed practice. Massing was most economical with the 4-unit, next with the 8-unit, and least with the 12-unit mazes. If this trend toward inferiority of massing with increasing size were a major factor in the increased superiority of massing with the spider maze, we would expect the 8- and 16-unit spider mazes to show less advantage for massing 4-unit U-mazes. But the contrary is the case. Not only is the economy of massing much greater with the spider maze, but comparison of the only two U-mazes and spider mazes having an equal number of units of true path, the 8-unit mazes, shows that the latter have a much greater superiority with massing than have the former. For example, there are only 28 percent more errors in distributed procedure with the 8-unit U mazes (**2**, p. 306), and about 120 percent more errors with the 8-unit spider mazes (Table I).

(*c*) It seems plausible, then, that the major difference between the U-maze and spider maze results is due to the fact that the latter have eight alternatives at each choice point, while the former have only two. This would support my hypothesis that forgetting the true path is one major factor of advantage in massed procedure, since the latter is penalized more heavily in the 8-choice than in the 2-choice situation.

(*d*) But in view of the role that practice played in a somewhat similar context (**4**) with the whole-part problem (increasing the relative economy of the part method with large mazes and decreasing it with small mazes), it would be desirable to have more data on the relation of transfer of learning from earlier mazes to massing and distribution. The spider maze experiment was indeed arranged with a view to making a preliminary attack on this problem, through a comparison of the 8-unit mazes learned at the beginning with those learned at the end of the experiment. In Table I this comparison shows that massing seems to lose relative to distribution with subject C and gain with subject W. C has five times as many errors (109/20) for distribution with the first four mazes, and only twice as many (21/10) with the last four mazes. On the contrary, W has about one and two-thirds as many errors (169/103) in distributed procedure with the first four mazes, and twice as many (57/28) in the last four mazes. Taken in conjunction with the fact that C makes only half as many errors as W in all mazes, we might find some slight ground for the hypothesis that, at the beginning of the experiment, eight units are most economical for C, but that at the end of the experiment she can handle 16 units with maximum efficiency in massed learning. On the other hand, the 8-unit patterns have not reached their maximum advantage with W at the end of the experiment.

It is quite clear, however, that since different mazes were learned by the two *Ss* in massed and distributed procedure, one cannot equate maze differences in massing and distribution for the first four or the last four mazes without combining the data from the two *Ss*. On the other hand, clarification of a complex pattern of variables cannot be expected to result from combining the results of *Ss* so different in ability, whether the differences in ability are due to practice or not. A further experiment is indicated.

Experiment II

A second experiment was carried on at the University of Toronto in 1941–42 with 18 honor psychology and graduate students as *Ss*. Each *S* was presented with the task of finding and memorizing the sequence of correct choices on eight 12-unit mental mazes. The *S* first read typewritten instructions, which were afterwards explained and illustrated by the *E*. *S* then called out numbers in order from 1 to 6, and *E* said 'right' as soon as *S* pronounced the correct number for a given choice point; whereupon *S* began once more calling out the numbers from 1 to 6. Thus, in the first trial on Maze I the dialogue went as follows, *S* saying the numbers and *E* saying 'right' at appropriate points: "1–2–3– (right), 1–2–3–4–5–6 (right), 1–2–3–4 (right)," etc. On subsequent trials *S* attempted to reproduce correct numbers only. If unable to recall a number for a particular choice point, *S* then called out numbers in any order he wished until *E* said 'right.' Trials were not timed, and within reasonable limits an *S* was allowed to proceed at his own pace. The criterion of learning was one errorless trial.

The 18 *Ss* were divided into four groups. Groups I and II (five *Ss* each) learned the mazes in the order 12345678, Groups III and IV (four *Ss* each) learned the mazes in the order 34127856. To avoid ambiguity, Arabic numbers are used as above to indicate specific maze patterns, and Roman numerals to indicate the position of a maze in the learning series. Thus, the average score of Maze I is the average of maze patterns 1 and 3 in position I. Groups I and III learned by massed practice (30 sec. interval between trials) and distributed practice (24 hour interval between trials) in the order MDDMMDDM, Groups II and IV used massed and distributed practice in the order DM MDDM MD. This arrangement counterbalanced practice effects and individual differences for each pair of mazes, and differences in maze difficulty for mazes I–II as compared with III–IV, and V–VI as compared with VII–VIII. The interval between mazes was never less than, and seldom more than, two days. Occasionally longer intervals were unavoidable, but were about equally divided between massing and distribution. *Ss* were instructed not to think of the maze patterns between trials.

Results

As in Experiment I, massed practice is throughout superior to distributed practice. With successive pairs of mazes the average errors in distributed practice are 110, 80, 52, and 42; in massed practice, 79, 31, 17, 17. In percentage terms the advantage of massing increases with each successive pair of mazes up to mazes V–VI. The percentage superiority of the 30-sec. over the 24-hour interval is: mazes I–II, 40 percent, mazes III–IV, 158 percent, mazes V–VI, 206 percent, mazes VII–VIII, 147 percent. The corresponding trial measures are: distributed practice, 9.6, 8.1, 6.2, 6.1 trials; massed practice, 9.0, 4.9, 3.6, 3.9 trials. Thus, with both trials and errors massed practice has the advantage. In percentage terms, distributed practice requires 6, 65, 72, and 57 percent more trials with successively learned pairs of mazes.

Using the F-test to determine the reliability of the differences between the corresponding means for massing and distribution, for 1 and 32 degrees of freedom Snedecor's tables give $F = 4.15$ at the five percent level, and $F = 7.50$ at the one percent level of significance (7). In the present experiment for massing-distribution differences with mazes I–II, III–IV, V–VI, and VII–VIII respectively, $F = 1.00$, 11.18, 13.60, 9.87 with the error measure, and 0.07, 5.11, 12.81, 6.70 with the trial measure. The error measures for each pair of the last three pairs of mazes and the trial measure for mazes V–VI are thus well below the one percent level of significance, while the trial measures for mazes III–IV and VII–VIII are between the one and five percent levels. On the other hand, we can place no dependence on the hypothesis that the massing-distribution differences for mazes I–II are due to anything other than random sampling.[2]

Discussion

Combining the presentation of readily forgotten (nonsense) materials with arrangement of conditions imposing a high penalty for forgetting is again associated with a great advantage for massed practice. Moreover, the superiority of massing is much greater for the mental maze than for the U-maze, in which only the former of these conditions obtains (2). With the mental maze, as with the spider maze, massing is superior to distribution in both errors and trials and throughout the learning, rather than only for errors in early trials, as with the U-maze. Moreover, the average superiority of massing for all conditions of the experiment (obtained by comparing total errors for all conditions) is about 100 percent for the mental maze and about 20 percent for the U-maze, and the greatest superiority of the former (mazes V–VI) is 206 percent, and for the latter (4-unit mazes) only 98 percent. The mental maze experiment therefore renders more plausible my hypothesis that the advantage of massing is increased by increasing the chances of error out of proportion to the amount of material traversed or reproduced in the completed learning act.

Regarding the role of transfer from maze to maze, there is no evidence that the rise in the relative advantage of massing from mazes III–IV to V–VI and the decrease from mazes V–VI to VII–VIII is reliable. On the other hand, the fact that between mazes I–II and III–IV the increase in relative advantage of massing is sufficiently great to shift F from almost complete non-significance to a high degree of significance indicates that massing is reliably less efficient with the first two mazes than it is with the remaining six.

[2] The writer is indebted to Dr. R. W. B. Jackson of the Bureau of Educational Research, University of Toronto, for setting up the statistical model and checking the results.

Since for both trials and errors the coefficient of variation $\left(\frac{100\sigma}{M}\right)$ is almost identical for mazes I–II and mazes III–IV, we cannot attribute the rise in the value of F with the latter pair of mazes to a decrease in the variability of the measures, and it accordingly seems reasonable to attribute it to the transfer of learning from mazes I–II to III–IV.

Suggestions for Further Work

The data seem fairly conclusive in regard to the relative gain of massed procedure from mazes I–II to III–IV, but it would be desirable to study further the effect of transfer between subsequently learned mazes. And in this connection, the suggestion in my results that the relation between the advantage of massing on the one hand and intermaze transfer on the other may be curvilinear has an important corollary for experimental design. For it should be noted that if the curve obtained by plotting massing-distribution differences against serial maze position is not a straight line, individual differences in stage of practice (inter-maze transfer) cannot be controlled merely by taking the average of a large number of *S*s. The reason for this may be clarified by a hypothetical example. Let us assume for purposes of illustration that massing is superior to distribution at only one stage of practice, and that at earlier and later stages massing and distribution show no differences in efficiency. Let us suppose further that the point at which massing surpasses distribution varies from individual to individual, either because of native ability or previous experience. Let us then take two hypothetical individuals A and B, and tabulate the (also hypothetical) number of errors on two mazes learned in succession.

	Maze I		Maze II	
	M	D	M	D
Subject A	40	80	30	30
Subject B	100	100	40	80
Totals	140	180	70	110

We have now on the average only 30 percent advantage (180/140–100) for massing in maze I and 56 percent in maze II (110/70–100), in spite of the fact that A actually has an equal number of errors for massing and distribution with maze I, but twice as many errors in distribution as in massing with maze II, *while the reverse is true of subject B*. We must of course assume that these are

the 'true' scores of A and B—that is, that random errors have been eliminated. That true scores cannot certainly be obtained does not affect the argument. The point is that unless the relation between massing-distribution differences and the position of mazes in the learning series is a straight line, averaging results from *Ss* differing in ability will tend to flatten the curve. I have of course taken an extreme case to highlight my point, but the principle is the same no matter how small the departure from linearity. Setting up a perfect experimental design would thus involve the impossible task of eliminating individual variability, but this need lead to no pessimism, since there are no perfect experiments. The solution most nearly ideal would be to run a series of experiments with groups differing in ability, but whose 'within group' variability had been reduced (by selection) to a minimum, and plot a curve showing the relation between serial maze position and massing-distribution differences for each group separately. It would then be possible to decide by inspection whether the different group curves represented fractional extents of a single function.

Summary

The relative economy of massing (30-sec. interval) and distribution (24-hour interval) of repetitions was investigated in two experiments with nonsense mazes, Experiment I with twelve 8-alley spider mazes (two *Ss*) and Experiment II with eight mental mazes requiring the selection and memorization of 12 digits each one of which is known to vary from 1 to 6 (18 *Ss*).

The marked superiority of massing over distribution found in both experiments was explained as follows. (*a*) With a 24-hour interval, successive trials strike the forgetting curve at a much lower point than is the case with a 30-second interval. (*b*) Six to eight choices at each choice point impose a high penalty for forgetting. This was supported by the finding that the present experiments showed massing more economical with all measures and throughout the learning, while an earlier experiment with two-choice U-mazes found massing only superior with errors and in early trials. (*c*) Following indications in previous work that internal interference increases with amount of material and decreases with number of mazes previously learned, the hypothesis that degree of internal interference favors distribution was investigated by varying the number of maze units (Experiment I) and by having *Ss* learn a series of mazes of the same length (Experiment II). However (possibly due to a non-linear relation between these variables and massing-distribution differences, in which case individual differences tend to flatten the curve), massing-distribution differences were not

found to vary with amount of material within the limits of the experiment, and position in the sequence of mazes learned only reliably increased the advantage of massing in Experiment II from mazes I–II to III–IV.

(Manuscript received February 28, 1944)

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THE THRESHOLD OF FLICKER FUSION AS A FUNCTION OF EXCITATION AND INHIBITION DUE TO CONDITIONING

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Pavlov's (**4**, **5**) theory of conditioning emphasizes two basic processes, those of excitation and inhibition, which are formed in the nervous system as a consequence of the establishment or extinction of a conditioned reflex. The excitatory process is produced during the formation of a conditioned reflex. The evidence for a state of excitation being present in the nervous system is based on the phenomenon that stimuli which were not used during conditioning are able to elicit the conditioned reflex. The excitatory process, formed largely in the sensory centers of the nervous system which receive the impulses of the conditioned stimulus, irradiates to adjacent areas, showing its maximum effects around the focus where it is formed, so that the most effective stimuli in eliciting the reflex are those which belong in the same sensory field as the original conditioned stimulus. The inhibitory process is formed when stimuli which originally tended to produce the conditioned reflex are no longer able to do so because of the establishment of a conditioned inhibition, or when the elicitation of the conditioned reflex is delayed. The inhibition may take place to the stimulus used originally as the conditioned stimulus, or to stimuli which have not been associated with the reflex but which, because of the process of irradiation, are able to elicit it. Once formed, the inhibitory process, like the excitatory, irradiates to adjacent areas of the brain.

The excitatory or inhibitory processes which Pavlov postulated were measured by their effects on conditioned and unconditioned reflexes, although the underlying assumption was made that the general reactivity of the organism was altered; in one instance a generalized state of excitation may take place, in the other, complete quiescence and sleep. If, indeed, some processes are formed in the central nervous system, whether or not they can be placed in the Pavlovian categories of inhibition or excitation, when a conditioned reflex is established or extinguished,[2] then the processes should have

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[2] Hilgard and Marquis (**3**) have summarized the arguments against the concepts of brain physiology formulated by Pavlov. A radically different explanation of conditioning is presented

a generalized effect in the locus of the central nervous system where they are produced. Should such a state of conditions be present, then it is logical to assume that not only should the motor responses which have connections in this or adjacent loci of the nervous system be affected, but other functions such as a sensory threshold whose central connections involve that particular part of the nervous system, should likewise be altered. The experiment reported here was based on these assumptions.

The fusion point of a pulsating light was used in the threshold determinations. Exploratory experiments on 14 Ss indicated that the average critical flicker frequency is decreased by 1.1 cycles if a patellar reflex is conditioned to a pulsating light and at the same time inhibited to a constant light, and is increased by 0.8 cycles if the opposite conditions hold. This problem was subjected to more rigorous experimental methods with the introduction of controls and is reported in the present study.

Methods and Subjects

The patellar reflex was elicited by a hammer weighing 274 gm. which was attached to a shaft of a pendulum. The distance of the hammer from the fulcrum varied from 56 to 58 cm. depending on the adjustment required for each *S*. The hammer was raised through a distance of 32 degrees and was released automatically by a cam mounted on a 110 volt A.C. motor. The blow was delivered to the patellar tendon of the right leg, the latter being placed on a stand and allowed to swing freely when the *S* was in a sitting posture. By the use of electrical markers, the time at which the conditioned and unconditioned stimuli were presented and (by a string and pulley attachment) the latency and amplitude of the reflex were recorded on a kymograph drum.

A ¼ watt neon lamp attached to a Clough-Brengle Oscillator, Model 79B, with a frequency range of 5 to 250 cycles, was used as the light source. The output of the oscillator was stepped up by a 90 volt D.C. battery placed in series with the lamp. A resistance of 0.1 megohm was placed in parallel with the output of the oscillator, and one of 150 ohms in series with the ground lead of the oscillator. The light was turned on automatically by a mercury switch mounted on a shaft which rested on the cam of the motor used in releasing the hammer which delivered the blow to the patellar tendon. The light lasted for one sec. and came 0.8 sec. before the blow to the patella was delivered. The *S* was seated 40 cm. from the light. The room was kept in semi-darkness, with an illumination of approximately 0.7 foot-candle.

The experimental procedure was as follows: Six preliminary measures of the patellar reflex and the point of flicker fusion were made separately. Ten thresholds of flicker fusion were then obtained in an alternating ascending and descending order. The *S* was then instructed that the starting of the motor was a warning that the light would go on. In order to have him attend to the light, he was to report whether or not the light was flickering. The light which was presented was either 10 cycles above or below the *S*'s fusion point as determined by the average of the 10 measurements. During the presentation of one of these stimuli the patellar reflex was elicited but not during the other. The stimuli were presented 100 times, and consisted of 50 pulsating and 50 constant lights. The order of these stimuli was the same for each *S*, but the sequence of presentation was originally decided upon by a chance selection. Immediately after the conditioning session 10 more thresholds of flicker fusion were obtained. A few reinforcements of the conditioned and unconditioned stimuli were then given, followed by a test for experimental extinction.

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by Guthrie (2). The present report does not attempt to deal with Pavlov's theories. Pavlov's concepts of excitation and inhibition as processes formed in the nervous system are used because the phenomenon reported here can be explained only by assuming that certain states are produced in the nervous system by a conditioned reflex and a conditioned inhibition.

The differences between the averages of the 10 determinations of the critical flicker frequencies before and after conditioning were used as an indication of the change taking place in the thresholds of flicker fusion.

Twelve male medical students, ranging in age from 23 to 26 years, were used as *Ss*.[3] The patellar reflex of six of the *Ss* was paired with the pulsating light and was not elicited when the constant light was presented. The order of conditioning and extinction of the reflex with the pulsating and constant lights was reversed for the remaing six *Ss*. The former group of individuals will be referred to as Group A and the latter as Group B.

Ten days after the first session (with the exception of one *S* who was tested two days later), Group B was conditioned in the same manner as Group A; the pulsating light acting as the conditioned stimulus and the constant light as the inhibitory stimulus.

Control experiments were instituted as follows: Group A, 10 days after the first session, was divided into two groups of three *Ss*. One of the groups received 50 pairings of the pulsating light with the patellar reflex, without any presentations of the constant light. The patellar reflex of the three other *Ss* was conditioned to a buzzer with the constant light acting as the inhibitory stimulus. The buzzer in the latter case performed the same function as the pulsating light in the first experiment of Group A. In these experiments the starting of the motor was used as a signal for the *S* to report whether or not the light was present. The reason for introducting these controls will become evident later.

Results

Table I shows the averages (with the variance of the distribution) of the 10 determinations of the flicker fusion thresholds before conditioning and the 10 determinations after conditioning, and the difference between the averages of each *S* of Group A, whose patellar reflexes were conditioned to a pulsating light of 10 cycles below the critical flicker frequency as determined before the conditioning session, but inhibited to a constant light of 10 cycles above the critical flicker frequency; of the first session of Group B, whose reflexes were conditioned to a constant light, but inhibited to a pulsating light; and of the second session of Group B, for whom the pulsating light served as the conditioned stimulus and the constant light as the inhibitory stimulus in the same manner as in the test of Group A. Whether a significant change of the critical flicker frequency occurred after conditioning was determined for each *S* separately and for the *Ss* as a group. In computing the values for each *S* the procedure for uncorrelated data was used (*cf.* Snedecor, **6**), since each threshold determination before conditioning could not be matched with each of the 10 determinations after conditioning. In the group comparisons, however, the differences between the averages

[3] Nine of the *Ss* were on diets deficient either in vitamin C, or in vitamins C and B, but since their results did not differ from those of the individuals who were on a normal diet, the vitamin deficiency appears to have had no influence on the results. Mention should also be made of the fact that weekly threshold determinations of the fusion point of a flickering light were made on these *Ss* for a period of seven months before the present experiment. They were also adapted to the conditioning apparatus, since the amplitude of their knee-jerk was tested a number of times prior to the experiment. The consistency of the results shown by these *Ss* can be accounted for by the absence of emotional disturbances produced by the experimental situations, since more recent experiments have shown that naive *Ss* not adapted to the experimental conditions are likely to give erratic responses, although the trend of their results is in agreement with those reported here.

TABLE I

RESULTS OF THE EXPERIMENT

Average critical flicker frequencies (Cff) before and after conditioning, the variance ($= \sigma^2 = \Sigma d^2/N - 1$, $N = 10$) of the distribution, and the differences between the first and last tests. The t-values for each individual and for the groups are given. Group A: patellar reflex conditioned to pulsating light and inhibited to constant light. Group B (first test): reflex conditioned to constant light and inhibited to pulsating light; (second test): same procedure as Group A.

Group A	1	2	3	4	5	6	t-values for groups
Cff before conditioning	42.2	40.2	41.0	46.6	42.2	40.4	
Variance	.18	0.18	1.56	0.71	0.62	0.27	
Cff after conditioning	39.7	38.9	38.9	44.6	39.9	38.7	
Variance	0.23	0.99	1.43	0.71	0.54	1.47	
Difference in Cff	2.5	1.3	2.1	2.0	2.3	1.7	11.4*
t-values (for individuals)	12.3*	3.8*	3.8*	5.3*	6.7*	4.0*	
Group B	**7**	**8**	**9**	**10**	**11**	**12**	
1st test							
Cff before conditioning	39.7	37.6	37.5	42.5	39.3	44.2	
Variance	1.34	0.93	4.94	2.06	0.23	0.40	
Cff after conditioning	41.0	36.9	38.7	43.4	39.3	42.7	
Variance	0.67	0.54	2.68	1.16	1.12	1.12	
Difference in Cff	−1.3	0.7	−1.2	−0.9	0.0	1.5	0.4
t-values (for individuals)	2.9*	1.8	1.4	1.6	0.0	3.8*	
2nd test							
Cff before conditioning	41.5	37.7	41.0	43.9	39.4	43.4	
Variance	0.94	0.46	3.78	0.54	0.49	1.16	
Cff after conditioning	40.4	37.2	39.7	42.1	37.5	42.4	
Variance	0.93	0.40	2.23	0.77	0.28	0.93	
Difference in Cff	1.1	0.5	1.3	1.8	1.9	1.0	6.0*
t-values (for individuals)	2.5**	1.7	1.6	5.0*	6.9*	2.2**	

*$P < .01$
**$P < .05$

of the pre- and post-conditioning tests were used to obtain the t-values, a procedure which takes into consideration the correlation between these averages. The t-values for each individual and for the groups are shown in the table.

The t-tests showed that the number of cycles by which the critical flicker frequency is decreased is statistically significant for Group A ($t = 11.4$, $P < .01$), and for the second test of Group B ($t = 6.0$, $P < .01$), for whom the pulsating light acts as a conditioned stimulus, and the constant light as an inhibitory stimulus. Individual comparisons show that a significant decrease of the critical flicker frequency was present in 10 of the 12 *S*s. If the reflex is conditioned to a constant light and inhibited to a pulsating light, as in the first conditioning session of Group B, no immediate change in the threshold of flicker fusion is detectable if the *S*s are compared as a group ($t = 0.4$, $P < .80$). Only one *S* in this group showed a

significant decrease of the critical flicker frequency and one a significant increase.

If the conditioning procedure was responsible for the decrease of the thresholds when the pulsating light is the conditioned stimulus and the constant light inhibitory, then it would be expected that if the order of presentation of the conditioned and inhibitory stimuli is reversed, the threshold should shift in the opposite direction and the critical flicker frequency of the fusion point should be increased. The fact that this result is not present for Group B as is seen from the results of the first conditioning session given in Table I, may mean, however, that it is not possible to augment the critical flicker frequency because under normal conditions the organism's maximum physiological limits are reached in making a discrimination. That this interpretation is correct is indicated by the results of the second conditioning test of Group B. Although there is a significant decrease of the critical flicker frequency on this test of Group B, the decrease is not as great as for Group A. The difference of 0.7 cycles between the averages of these two scores is statistically significant ($t = 2.5$, $P < .05$). The conditions under which Group A was tested and Group B was retested were similar, so that the difference between their thresholds can be accounted for only by assuming that the first conditioning test of Group B set up some process which tended to raise the critical flicker frequency, but which could not be detected in the threshold determinations because the first determinations were the maximum which could be obtained under any conditions. If this process was still present on the second test, then the critical flicker frequency could not be lowered to as great an extent as in Group A, who had not been exposed to the first conditioning test. In this connection, the results of *S* No. 8 (Table I) of Group B are of interest. He was tested the second time only two days after the first test whereas the other five *S*s were retested 10 days later. The decrease of his fusion point was less than that of the other five *S*s and was not statistically significant. (The only other *S* in this group who did not show a significant decrease was No. 9, whose variance was very high.) If the first test created some process tending to augment the fusion point, and if some time is necessary to dissipate this process, then one would expect that after two days the effects of the first session would be more pronounced than after 10 days, so that the fusion point would be less likely to be depressed by the second test.

The results of Groups A and B also show that the decrease of the point of fusion cannot be explained in terms of such factors as sensory adaptation, changes in muscle tonus as the experiment progressed, etc., but must be due to some process in the central nervous system, because if the former explanation were true, then

the flicker fusion thresholds would be similar for Group A and the two sessions of Group B, since in all experiments the above factors were kept constant and only the nature of the conditioned and unconditioned stimuli was varied.

Two conditions, that of a tendency to form a conditioned reflex to a flickering light, and to inhibit it to a constant light, were present in the situation which served to decrease the threshold of flicker fusion. The next question to be answered was whether either condition alone would depress the threshold in the same manner as when both of them are present. In order to obtain data on this problem, the patellar reflexes of three individuals of Group A (Nos. 1 to 3, Table I) were conditioned to a pulsating light, the constant light being entirely absent; the reflexes of the other three *Ss* were conditioned to a buzzer, the constant light acting as an inhibitory stimulus. In the first group, only the positive conditioned reflex from the original conditioning situation is used; in the second group, only the inhibition to a constant light is established without the reflex being conditioned to the pulsating light. In order to keep the number of excitatory and inhibitory stimuli the same as in the first conditioning session, there were 50 pairings of the reflex with the pulsating light in the first group of *Ss* and 50 presentations of the constant light without the elicitation of the reflex in the second group. The average decrease in the critical flicker frequency was 0.5 cycles when only the pulsating light was used as the conditioned stimulus without the constant light being introduced, and 0.6 cycles when an inhibition was established to the constant light with the absence of conditioning to the pulsating light. These results compared with the average decrease of 2.0 cycles for each group of three *Ss* when both pulsating and constant lights are used in conditioning, show that neither the stimulus which is conditioned to the reflex nor the stimulus which inhibits the reflex when used alone is able to depress the fusion point in the same manner as when both of the methods are combined.

Conditioned reflexes appeared in two *Ss* during the test for experimental extinction at the conclusion of the experiment, and the tendency to give conditioned responses by many of the *Ss* was shown by the shortening of the latency of the reflex. These were not of sufficient number, however, to make comparisons between the strength of conditioning and the change in the critical flicker frequency.

Discussion

A patellar reflex conditioned to a pulsating light will tend to be elicited by a constant light because all stimuli in the same receptive field as the original conditioned stimulus will have this property.

This tendency for the constant light to elicit the reflex is inhibited by introducing the constant light without any reinforcements of the reflex. Two stimuli, one below the critical flicker frequency and one above, are used to condition and to inhibit the patellar reflex. The reverse order holds if the reflex is paired with the constant light and inhibited to the pulsating light.

The stimuli producing the conditioned reflex and the inhibition must have an additive effect in altering the threshold of flicker fusion, since a pulsating light as a conditioned stimulus and a constant light as an inhibitory stimulus must be present to depress the point of fusion, and reversing these conditions will produce a tendency to augment the fusion point. The additive effects of excitation and inhibition are opposed to the Pavlovian thesis that these processes produce opposite results in the nervous system, although his conclusions were based on the manner in which motor responses are affected and not sensory thresholds. Pavlov (4) did show, however, that an excitatory state may eventually change into an inhibition without the introduction of an inhibitory stimulus, or the inhibition may lead to a greater state of excitation. Since similar conditions may produce the two opposite processes it is to be concluded that intimate relationships exist between them.

The processes responsible for the altered threshold of flicker fusion must be present in the sensory area of the nervous system of the impulses produced by the conditioned inhibitory and excitatory stimuli, because the central connections used in determining the flicker fusion threshold would lie in this area, and not in the sensory or motor areas of the impulses of the unconditioned stimulus. The nature of the processes is open to conjecture.

If the processes which alter the critical flicker frequency are due to some condition in the central nervous system, then the further assumption must be made that the fusion of a pulsating light is a central phenomenon, and not due to the lag of photochemical reactions of the retina as has been suggested. Bartley (1) has already presented data on flicker fusion which cannot be accounted for by postulating that the fusion occurs in the retina. A more direct attack on this problem was performed by Walker, Woolf, Halstead, and Case (7), who picked up action potentials from different parts of the brain of the monkey when the eyes were stimulated by a pulsating light. They found that the action potentials of the cortex of the striate area fuse at about the threshold point of the animal's ability to discriminate, whereas other parts of the brain, such as the geniculate bodies, show no fusion of the action potentials at frequencies above the animal's critical fusion point, thus indicating that fusion is a cortical phenomenon. On the basis of this evidence,

the hypothesis that the conditions of the present experiment produced some change in the striate area of the cortex, thus affecting the flicker fusion threshold, should be considered as a possibility.

Summary

1. If a pulsating light 10 cycles below the individual's point of fusion is paired with the patellar reflex and interspersed with a constant light of 10 cycles above the threshold of fusion at which time the reflex is not elicited, a significant decrease of the number of cycles at which fusion takes place will occur upon termination of the experiment.

2. If the constant light is paired with the reflex and the pulsating light is not, no change in the threshold of flicker fusion will take place immediately after the experiment is terminated. A tendency to raise the fusion point will be evident from the fact that if, later, the pulsating light is paired with the patellar reflex and the constant light is used as the inhibitory stimulus, the fusion point will be lowered, but not to as great an extent as in *Ss* who were not exposed to the first conditioning session.

3. These results can be accounted for by assuming that some processes are established by the impulses of the conditioned and inhibitory stimuli in the sensory areas of the central nervous system. The processes produce a tendency to augment or depress the critical flicker frequency.

4. It is necessary to have a conditioned reflex and an inhibition established at the same time in order that the tendency to depress or augment the fusion point be present, since using only the conditioned stimulus or the inhibitory stimulus produces little change in the threshold. This is taken to mean that the processes of excitation and inhibition may have an additive effect in either lowering or raising the fusion point, rather than opposite effects noted on conditioned reflexes.

(Manuscript received March 17, 1944)

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